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APRIL 1953
VOLUME 57 NUMBER 4

Published Monthly by
AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $12.00

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Communications regarding subscriptions, reprints, etc., should be addressed, A. M. A. ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

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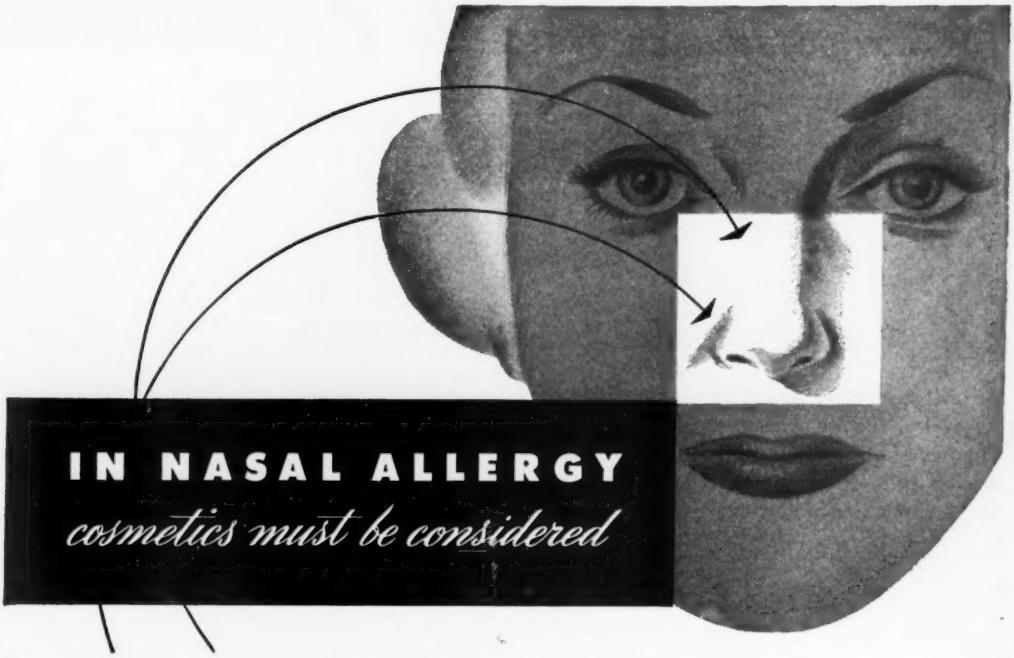

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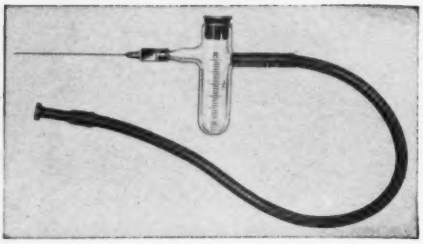

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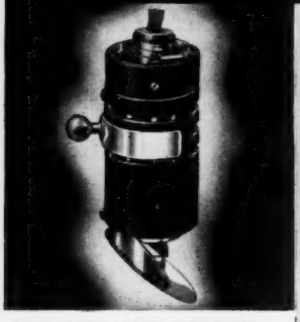

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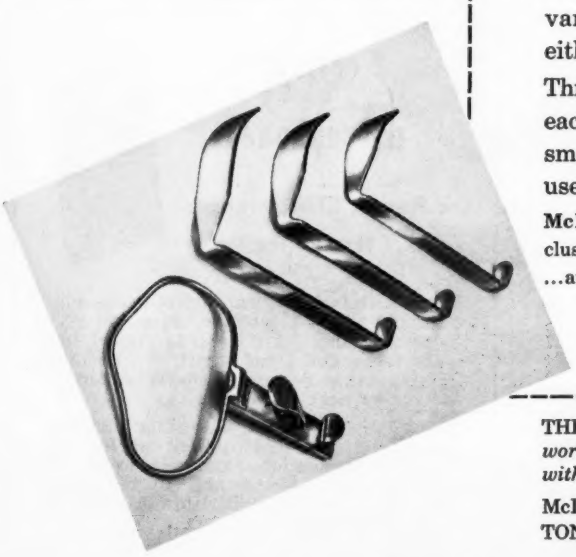

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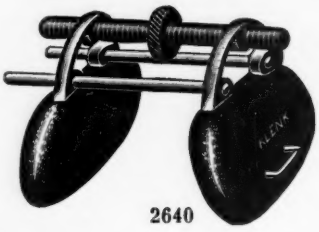

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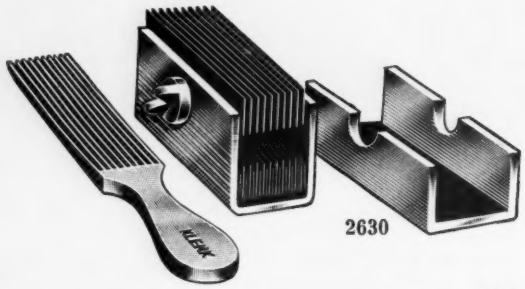

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CARDIAC ARREST FROM THE OTOLARYNGOLOGIST'S VIEWPOINT

R. M. HOSLER, M.D.
CLEVELAND

THE INCIDENCE of cardiac arrest during otolaryngological operations appears higher than would be casually anticipated. Considering the number of such operations carried out each year, the relative incidence is probably no higher than in other fields. However, the number of cases with successful resuscitation in the past, has been small. This is understandable in such specialties as represented by otolaryngology, and it is in such specialties that cardiac arrest is harvesting a large toll. Usually the otolaryngologist has had meager training in general or thoracic surgery, or it has been a considerable length of time since he has observed this type of surgery. With this background—and heaped upon it the sudden realization of cardiac arrest—the otolaryngological surgeon is inhibited from slitting open the chest.

In this specialty, the relatively frequent occurrence of cardiac arrest in young people and in persons whose hearts are, for all intents and purposes, normal, is alarming and somewhat mystifying. When this occurs in a person with a so-called normal heart and a normal pair of lungs it can be compared to turning off the ignition key in an expensive automobile. Unless the ignition key can be turned on again, the car is as worthless as a pile of junk. Unless practical resuscitative principles are instantly carried out, the patient will remain dead.

This short article will attempt to enumerate and illustrate some of the practical aspects of the prevention and treatment of this catastrophe. This subject is not a new entity, but few of us became aware of this entity during our formal medical education.

A specialist rating in this particular field is obtained after special training, which, among other specific things, prepares him to successfully handle any emergency or eventuality in that field—hemorrhage, etc. By the same token, he must through previous preparation be able to treat intelligently cardiac arrest when it occurs. Is he or has he been trained or fully prepared for this eventuality? A surgeon is unable to get ready for any emergency in a moment. His actions are guided and influenced by years of careful preparation.

Education in this subject has been neglected, and today, after a hundred years of anesthesia, we find the subject of cardiac arrest still begging for recognition. A curious lack of information seems to have prevailed through all these years. The condition it describes and its connotations are intimately associated with anesthesia. The first case of cardiac stoppage occurred in 1848 in a 15-year-old girl while under chloroform anesthesia for the removal of an infected toe nail.

Sudden cessation of the heart beat in the operating room is the enigma and challenge of present-day surgery and anesthesia. It can occur under local anesthesia as well as under general anesthesia. It can happen to the accomplished surgeon as well

as to the intern. Furthermore, it seems to take place as frequently during a simple operation as during a complicated one. It occurs as frequently in the young as in the old. It haunts all hospitals.

Reliable past and present-day figures on the incidence of this catastrophe are nowhere available. However, it is my considered opinion that it is occurring more frequently than in the past. A conservative figure that is often used is that cardiac arrest will occur on the average of five times each year in every active 700 bed hospital.

The previous smug, self-satisfied attitude that "this will never happen to me" can no longer be tolerated. It is only natural that one have this complacent attitude. It is only after disaster has struck that we acquire humility. This humility then becomes the offspring of knowledge.

In taking into consideration the etiological factors in this disaster it fundamentally resolves itself into a lack of oxygen in the vital tissues. There is a breakdown of the oxygen system. In some instances the specific factor can be pinpointed; however in others it can be likened to a chain of circumstances, and somewhere a link is broken which precipitates this disaster.

Briefly, some of the predisposing factors [1] are position of patient, decreased vital capacity, tracheal obstruction, poor oxygenation, anemia, anxiety, insufficient atropine, shock, poor cardiac filling, torsion of the heart, excessive accumulation of carbon dioxide in spite of good oxygenation, reflexes, errors of judgment, excessive numbers and quantities of preanesthetic and anesthetic drugs, apathy, and cardiac disease. In general, it can too often be said that the patient was overdrugged and underoxygenated.

Obviously the best treatment for this condition is its prevention. Overemphasis of cardiac resuscitation, if the former were neglected, could be likened to locking the barn after the horse had been stolen. However, if a surgeon is confronted with this extraordinary situation and hopes to be successful, he must have a definite plan of attack indelibly stamped into his brain, so that it may be called upon almost as a reflex. It is well to remember that once a proper reflex has been established it means action without error. There is no time to improvise, there is no time to call a consultant, and there is no time for erroneous action. There is a time limitation of three to five minutes which must be overcome; otherwise the upper centers of the brain will be irreversibly damaged.

When does one institute resuscitative measures? In other words, how do we know the patient is dead? Ordinarily this is not difficult for the surgeon and anesthetist to conclude. If there is no palpable pulse, no recordable blood pressure, and no respiration, the patient for all intents and purposes is dead, and if nothing constructive is done, will remain so. Often the surgeon is in a position to palpate directly one of the major arteries.

RESUSCITATION PROCEDURE

The resuscitation procedure is separated into two distinct steps. This is a basic contribution to resuscitation because it separates the things to do and things not to do. It focuses attention on the oxygen system and not on the heart beat. It highlights the basic problem, which is to keep the brain viable.

1. Hosler, R. M.: A Manual on Cardiac Resuscitation, Springfield, Ill., Charles C Thomas, Publisher, 1953.

The two components are (1) reestablishment of the oxygen system, and (2) restoration of the heart beat. These are distinct steps, and one should not be confused with the other. With an understanding of these, the problem becomes relatively simple, provided that one can place and keep first things first and can exert the necessary perseverance.

Once the emergency has arisen there is no time for indecision, hesitation, or vacillation. It is just as important to know what not to do as what to do. It seems that the unprepared surgeon will take time to carry out these unnecessary steps in order to convince the operating team that the thoracotomy is the last resort. In the meantime precious moments are consumed while a life is hanging in the balance.

Do not take time (1) to adjust the blood pressure cuff; (2) to listen for the faint heart sounds; (3) to give "old-fashioned" artificial respiration; (4) to inject drugs through the chest wall; (5) to call for a transfusion, (6) to call for an electrocardiogram. The time taken to carry out such maneuvers will insure against success in almost all cases.

I. Reestablishment of the Oxygen System (The Emergency Act).—The first step is for an alert anesthetist to place a tube into the trachea and deliver 100% oxygen into the lungs under positive pressure. Once the anesthetist can adequately aerate the lungs, the surgeon proceeds boldly to open the left chest [2] and squeezes the heart and intact pericardium upward against the sternum. The crisis is now over. Oxygenated blood is being delivered manually to the brain by the hydrodynamic pump. The brain can now be kept viable for an indefinite period. If the heart at this stage is not beating rhythmically, the next step is to insert a large rib retractor and quickly open the pericardium and immediately resume massage. Once the pericardium is open, massage can be carried out with either two hands or one and a more effective circulation can be obtained. In the meantime the anesthetist has lowered the table to about 10 degrees in the Trendelenburg position.

II. Restoration of the Heart Beat.—The surgeon may be confronted with either one or two conditions of cardiac arrest in the operating room, namely, cardiac standstill and ventricular fibrillation. The incidence of finding the first condition is, fortunately, greater, and this condition is the easier of the two to treat. Up to this juncture it is not known whether or not the heart is in standstill or ventricular fibrillation. Since the procedure is entirely different in the two conditions, the surgeon now stops massage momentarily and observes the ventricles. If there are no fibrillary movements he can assume that the ventricles are in a state of asystole.

A. Method of Restoring Beat in Cardiac Standstill: The heart in asystole may start beating after hand massage alone. If after a considerable period of adequate massage it should not start, about 4 to 5 cc. of 1:10,000 epinephrine solution is injected into the chamber of the right ventricle and massage is continued. The heart will then begin to beat if all conditions are favorable. In difficult and prolonged cases a mechanical respirator of the Rand-Wolfe [3] type is of great assistance.

B. Method of Restoring Beat in Ventricular Fibrillation: The problem of overcoming ventricular fibrillation (which is truly a convulsion and purposeless movement of cardiac muscle) is a more serious and usually more difficult one. There is

2. The fifth left interspace is preferred.

3. Wolfe, K., and Rand, H. J., III: Electro-Mechanical Aids in Resuscitation and Anesthesia, Ohio M. J. **46**:39, 1950.

such incoordination of cardiac muscle fibers that the blood pressure is not maintained. The brain must be kept viable and the heart muscle a pink color by effective manual massage. An electric shock is usually required at this juncture. The large suction-cup electrodes are applied, and the shock is furnished by a defibrillating machine.[4] We use the practical and fool-proof Beck-Rand Defibrillator. Once the heart has been restored to a coordinated beat, the remaining procedure is similar to that of standstill. In the difficult case 4 to 5 cc. of 1% procaine hydrochloride injected into the chambers is a valuable aid. It may be necessary to repeat the electrical shock several times before the heart returns to a normal beat. The secret of success in these instances is to have the body and heart muscle in a good state of oxygenation. This is carried out by good excursion of the lungs and by adequate cardiac massage. Attention must be directed to obtaining good emptying of the right ventricle as well as the left ventricle in order to sustain an effective circulation.

The major causes for failure to restore the heart beat are (1) poor aeration of the lungs, (2) ineffectual manual massage, (3) retention of carbon dioxide, and (4) intrinsic heart disease.

An approach to this subject is not complete without a brief discussion of drugs which may be of particular value in this emergency. If the chest is opened immediately after the onset of the catastrophe and if the proper steps are carried out, drugs are not needed as a general rule in either standstill or ventricular fibrillation. However, in the difficult case it is well to know that there is a definite place for procaine and epinephrine. Do not clutter up the mind or the operating table with other drugs at this time.

In the successful case, do not be in a hurry to close the chest. Observe the heart beat for some time before the decision is made. The patient should be kept on the surgical floor until you are entirely satisfied. Fluids and transfusion may judiciously be given.

Operations should not be undertaken unless the anesthetist has intratracheal tubes, oxygen, and a rubber bag readily at hand. Each operating floor should have a sterile emergency resuscitation kit and a defibrillating machine readily available.

FACTORS RELATED TO CARDIAC ARREST

A review and discussion of some of the factors pertaining to cardiac arrest in otolaryngology may be a helpful exercise.

Anesthetic [5] and preanesthetic drugs can be potent factors. A death from anesthesia excites much comment from the laity, while a direct surgical complication will create less discussion. Some of the significant actions of the ordinary drugs in this group bear repetition. The accepted figure for the mortality [6] from ether anesthesia in skilled hands is 1 in 16,000. Chloroform [7] is 25 times as toxic to the myocardium as ether.

4. Hosler, R. M.: The Emergency Treatment of Cardiac Arrest, J. Am. A. Nurse Anesth. **20**:18, 1952.

5. Oliver Wendell Holmes applied the name to the state produced by ether in the hands of Morton.

6. Stavraky, G. W.: Effect of Amytal on Autonomic Nervous System as Indicated by Salivary Glands, J. Pharmacol. & Exper. Therap. **43**:499, 1931.

7. Salter, W. T.: Textbook of Pharmacology, Philadelphia, W. B. Saunders Company, 1952, p. 167.

Procaine has a depressant effect upon the heart and can stimulate the brain to convulsions. The excitatory phase can be antagonized by barbiturates. If the rate of absorption of the local anesthetic exceeds the rate of detoxification, the concentration in the blood mounts. This may quickly proceed to toxic levels and rapidly jeopardize a surgeon's practice. When the drug is injected into a mucous membrane, it is absorbed almost as quickly as if it were administered intravenously, since the mucous membrane has a rich blood supply. The heart rate will be slow and weak from overdosage of procaine or cocaine. It depresses the myocardium and the conduction system. The heart may actually stop within a few minutes. Collapse may be more sudden with cocaine, and the initial difference is that it may be so rapid that a stage of nausea does not occur. Deaths from cocaine have been reported from doses as low as 20 mg. Doses as high as 200 mg. of procaine hydrochloride should be avoided. A fatality may occur when least expected.

Epinephrine causes the general rate of oxidation of all tissues to increase; therefore there is a greater need for oxygen. This need is hard to accommodate in the myocardium which is jeopardized by coronary sclerosis. An irritable heart or one under the influence of an anesthetic may be the seat of sudden precipitation of ventricular fibrillation by this additional action of epinephrine. The drug dibenamine[8] blocks the effect of adrenergic principles and can eliminate the arrhythmias due to epinephrine.

Atropine will block the effect of acetylcholine. The latter substance is thought to be produced by stimulation of the vagus nerves. Its action is brief as it is highly unstable, but over the course of a few minutes it may produce dramatic effects. The effect on the heart is to cause a decrease in the rate and force of contraction. This may go on to complete heart block. Quinidine[8] will prevent this bradycardia. Acetylcholine is unstable in the presence of the oxidase cholinesterase. The latter is thought to be present on the surface of the red blood cell. Consequently another need for correcting an anemia is found. In their action on the central nervous system and cardiac protective mechanism, atropine and scopolamine differ appreciably.

Let us consider the unfortunate events in a hypothetical case. The patient is a well developed adult with a normal heart and a normal pair of lungs who is about to undergo a tonsillectomy with local anesthesia. He may or may not have had any preoperative medication. The up-to-date operating room consists of a small confining cubicle. The patient is seated in a rigid, very straight-backed chair. Cocaine is applied to the area followed by the injection of 2% procaine hydrochloride.[9] The rate of absorption of the procaine in the richly vascular area greatly exceeds its detoxification rate, and temporarily there is an overdose of the drug in the blood stream. The peripheral effects cause a dilatation of the vessels in the extremities. Shortly thereafter the patient becomes pale, clammy, apprehensive, and nauseated. A sinister bradycardia develops. This is the effect of an overdose of the agent and it may be quickly followed by sudden cardiovascular collapse and death. What is the mechanism of this sudden death? It is felt that this is the effect of a high concentration of procaine (transitory overdosage) in the blood stream and not hypersensitivity. This drug quickly reaches the heart. Its effect on the heart is to relax its tone and cause a series of weak beats. Now if the cardiac filling is also poor,

8. Salter,[7] p. 757.

9. A 1% solution is preferred.

owing to position and peripheral vascular collapse, within a dozen or so beats the coronary pressure can become ineffective and cardiac arrest quickly and unbelievably supervenes.

Briefly, what should the alert surgeon attempt to do in such a situation? Provided the patient is not yet pulseless, he should be placed in such a position that he is practically standing on his head. This will give him a transfusion of 600 to 700 cc. of whole blood which has been stagnant in the dependent parts. How can a matched transfusion be given any more quickly? Next the respiration must be maintained by mechanical methods. These maneuvers may be enough to prevent the termination in cardiac arrest, since within a few minutes this transitory overdosage will have an opportunity to be eliminated or detoxified. If cardiac arrest has occurred, the above measures are carried out and without hesitation the chest is boldly opened and cardiac massage instituted.

I feel that one of the gravest hazards in otolaryngological operations is the sitting position. The chair should be easily adjustable, as is a dental chair, so that the patient may be virtually stood on his head if necessary. As doctors and specialists we have overlooked the first lesson we learned in medical school, the care of a patient who has fainted. We first must call Sir Isaac Newton into consultation.

REPORT OF A CASE

The following is a summary of a case report forwarded to me by another physician.

A 5-year-old patient had a tonsillectomy and adenoidectomy performed under general anesthesia. The child made an uneventful recovery. Five days later she returned in the morning complaining of bleeding of the nose of four hours' duration. She seemed to be in good shape and had not lost too much blood. Epinephrine packs were inserted into the nose, and the patient was put to bed in the hospital. At 9:30 that morning through consultation it was decided that the bleeding was coming from the adenoid area, and it was thought advisable to reanesthetize the patient and pack the adenoid area. Atropine, 1/300 grain (0.20 mg.), was given 15 minutes before the beginning of the anesthesia with nitrous oxide and oxygen. About the time the patient was considered to be under surgical anesthesia, there was some gurgling in the throat. A mouth gag was inserted in the mouth and a large clot had slipped down from the adenoid area into the region of the epiglottis. This clot was removed quickly with a sponge forceps. The patient began to wake up and in the meantime became cyanotic. Palpation of the heart over the chest area showed it to be very slow, and it beat only a few times before it ceased beating. Oxygen was immediately applied by compression upon the oxygen bag. After three minutes by the clock the chest was entered and the heart was at complete standstill. The pericardium was opened and the heart was massaged manually, and 0.5 cm. of epinephrine hydrochloride was injected into the right heart chamber. The heart was massaged for 45 minutes without response.[10] Upon opening the chest the left lung was completely collapsed. There was no method available for intratracheal intubation. Some of the oxygen pumped through the bag entered the stomach.

In 1950 Beck, Rand, and I instituted a practical course in cardiac resuscitation. It is a two-day course and is held once a month in Cleveland. Applications are handled by the Cleveland Heart Society at 2073 East Ninth Street, Cleveland. The course has been enthusiastically received, and to date over 600 persons have enrolled

10. Failure was probably due to inability to carry out adequate oxygenation. If intubation was not possible, tracheotomy and the introduction of a rubber tube might have been beneficial. There also may have been blockage of the left mainstem bronchus by clots.

from the four corners of the United States and some foreign countries. In October, 1952, two courses were given at the Army Post-Graduate School at the Walter Reed Hospital for 70 participants.

Planning for the eventuality of cardiac arrest is one of the best ways to prevent its occurrence. An exact pattern must be followed to insure success. The planning must be on an individual as well as on an institutional plane, if better results are to be obtained. Preparation will save lives and time-consuming indecisions.

Is it demanding too much that all surgeons and all institutions be prepared to take care of this emergency efficiently? The primary question today is: Should the average surgeon be prepared to carry out resuscitative methods in his community? Should each hospital have the necessary training program and the necessary special equipment? That all surgeons have the knowledge to carry out the resuscitative procedure and that all hospitals have the necessary equipment—these are the goals for which we strive.

SUMMARY

An approach is made to the treatment of cardiac arrest. A brief review of the pharmacology of certain drugs used by the otolaryngologist is undertaken. Some of the causes of cardiac arrest are illustrated and discussed.

PATHOGENESIS AND HISTOPATHOLOGY OF CHRONIC ADHESIVE OTITIS

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SOME FACTS speak in favor of the assumption that in present-day otology chronic adhesive otitis, instead of manifest suppurative chronic otitis, will become the commonest inflammatory disease of the middle ear leading to serious deterioration of hearing. Thanks to modern antibacterial agents, it seems possible that development of manifest suppurative chronic otitis can in most cases be prevented by proper therapy. In spite of the new drugs, prevention of chronic adhesive otitis can hardly be equally successful, owing to the somewhat obscure origin and insidious development of this disease. On the contrary, there is reason to believe that some conditions which previously would have resulted in manifest suppurative chronic otitis may now develop into chronic adhesive otitis. It thus seems that a reconsideration of chronic adhesive otitis is appropriate.

As regards clinical characteristics, there is a close resemblance between chronic adhesive otitis and otosclerosis. Probably some of the lesions diagnosed as otosclerosis actually have been instances of chronic adhesive otitis. This question has received special attention in the most recent clinical investigation of adhesive otitis (Lumio[1]). From the point of view of differential diagnosis the following features of this disease are particularly stressed:

Chronic adhesive otitis may start at any age, frequently in childhood. There seems to be a certain hereditary tendency, though it is not so distinct as in otosclerosis. Often the history reveals that the patient has earlier suffered from one or several middle ear inflammations, usually mild, that have received inadequate treatment or none; paracentesis especially is frequently omitted. Inflammatory processes in the nose and in the nasopharynx and adenoids and other pathological conditions leading to Eustachian tube obstruction are fairly common. The tympanic membrane shows no perforation but almost invariably pathological alterations. Generally the mastoid bone is scantily pneumatized. The hearing loss is of the same type as in otosclerosis, though in adhesive otitis features indicating inner ear deafness are often noted at an early stage. The hearing deteriorates steadily, or by degrees, with stationary intervals of varying length. The disease may end in complete deafness. Tinnitus is a common symptom. Occasionally there is also aural vertigo, especially during head colds and in connection with inflation of the middle ear. In some cases good improvement has been obtained by fenestration. Other therapeutical measures are usually without effect.

From the Department of Pathological Anatomy, University of Helsinki, Prof. Dr. Arno Saxén, Director.

1. Lumio, J. S.: Contribution to the Knowledge of Chronic Adhesive Otitis: The Diagnosis, Acta oto-laryng. **39**:196, 1951.

The recent literature contains no studies dealing with the histopathology and the pathogenesis of chronic adhesive otitis. The textbooks and manuals of otology [2] base their descriptions chiefly on studies carried out at the end of the last century and during the first decades of the present century. The diversity of opinions is reflected, for instance, in the numerous designations used: adhesive otitis media, adhesive process, regressive process, chronic catarrh of the middle ear, dry catarrh of the middle ear, proliferous inflammation of the middle ear, etc. Mostly, adhesive otitis is distinguished from otitic residua, the latter being a stationary condition, while adhesive otitis is marked by a progressive deterioration.

An earlier study of mine [3] on the pneumatization of the middle ear contains some observations regarding the pathogenesis and histopathology of chronic adhesive otitis. Because these problems seem to be of current interest, it may be appropriate to study them more closely. Particular attention will be paid to the following aspects: 1. In what kind of conditions does extensive organization of exudate occur in the middle ear? 2. Under what conditions do the resulting adhesions develop into chronic adhesive otitis? 3. What theoretical conclusions can be drawn regarding the present-day prophylaxis and treatment of adhesive otitis?

MATERIAL

The present material is mainly the same as that used in my study on mastoid air cell formation.[3] A detailed description of the material and of the histological technique will be found in the publication mentioned. The essential part of the material consists of 89 histological series of temporal bones which Professor Saxén supplied from his collections. The past histories had been carefully recorded, and thorough otologic examinations including otoscopy and hearing and labyrinthine tests repeated at intervals of three to six months until the patient's death had been made in all cases. In 13 cases the clinical and histological findings suggested adhesive otitis. The remainder, except two cases without any signs of previous or active inflammation, were cases of otitic residua of different type or acute otitis of varying severity. Unfortunately, all the patients were aged persons, over 70 years on the average. Therefore, it was impossible to evaluate with certainty to what extent pathological changes in the middle ear were responsible for the hearing loss observed in hearing tests.

An additional series comprising 12 serially sectioned temporal bones of adults, aged from 19 to 60 years, was collected by me from the department of pathological anatomy of Helsinki University. Only one of these cases was clinically studied; the others were selected on the basis of postmortem otoscopic findings indicating acute otitis or otitic residua.

A series comprising 15 temporal bones of newborn infants was used for studying the importance of foreign body otitis. Unfortunately, no bacteriological examination had been made, the series not having been originally collected for this purpose. All infants had died during delivery or a few hours after from causes other than infection, as confirmed by autopsy. Thus, it may be assumed that the foreign body otitis was sterile.

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2. (*a*) Walb, H.: Krankheiten der Paukenhöhle, in Schwartze, H.: Handbuch der Ohrenheilkunde, Leipzig, F. C. W. Vogel, 1892, Vol. 2, pp. 183-195 and 280-289. (*b*) Eschweiler, R.: Rückstände und Verwachsungen in der Paukenhöhle, in Handbuch der Hals- Nasen- und Ohrenheilkunde, edited by Denker, A., and Kahler, O., Springer-Verlag, 1926, Vol. 7, pp. 393-408. (*c*) Wittmaack, K.: Der Adhaesivprocess, in Handbuch der speziellen pathologischen Anatomie und Histologie, edited by Henke, F., and Lubarsch, O., Berlin, Springer-Verlag, 1926, Vol. 12, pp. 406-414. (*d*) Ballenger, W. L.; Ballenger, H. C., and Ballenger, J. J.: Diseases of the Nose, Throat, and Ear, Ed. 9, London, Henry Kimpton, 1947, p. 640. (*e*) Fowler, E. P.: Medicine of the Ear, New York, Thos. Nelson & Sons, 1939, p. 160.

3. Ojala, L.: Contribution to the Physiology and Pathology of Mastoid Air Cell Formation: Histological Studies of Aged Individuals and Newborn Infants, Acta oto-laryng., Supp. 86, p. 1, 1950.

PATHOLOGY OF THE ORGANIZATION PROCESS

Organization of exudate is a complicated occurrence which presupposes the combination of several factors. This phenomenon occurs most frequently in cavities lined by endothelium or endothelium-like epithelium. The latter group is represented by the middle ear cavities, in particular the air cells. Perhaps the pulmonary alveoli are most closely comparable with them. Organization of exudate occurs seldom or never, except after necrotizing inflammations, in cavities lined by differentiated mucous membrane, such as the Eustachian tube or the nasal accessory sinuses. If a comparison with general pathology is desired, pneumonia may be the most suitable parallel to middle ear inflammation. It is possible that the organization of exudate in these two diseases is due to similar factors. Many problems involved are beyond the scope of a single histological study, and the purpose of the following discussion, which is based on previous investigations and my own observations, is chiefly to give some ideas for further studies.

Several authors (e. g., Krainz,[4] Lange,[5] Singer[6]) consider that only exudate containing fibrin undergoes organization. Döderlein,[7] on the other hand, has found organization also in afibrinous exudate, provided that it contains inflammatory cells. Wittmaack[8] holds the view that foreign body otitis in infancy is the most important cause of organization processes.

All specimens from the newborn infants in the present material showed foreign body otitis, probably sterile, of varying severity. The mucous membrane was more or less infiltrated by inflammatory cells. The epithelium was always intact. In addition to the solid particles of amniotic fluid, the secretion in the middle ear cavities contained numerous pus cells but no fibrin. In none of the cases were there any signs of incipient organization.

Among the adult specimens there were several showing mild otitis. The mucous membrane of the middle ear was edematous and hyperemic and showed slight infiltration with inflammatory cells. The epithelium was intact. In the middle ear cavities there was afibrinous serous or mucous secretion containing varying numbers of inflammatory cells. There were no signs indicating organization of the exudate.

The present series also included several specimens with evidence of severer inflammation. The mucous membrane of the middle ear was intensely infiltrated with inflammatory cells, and the lining epithelium was destroyed in areas of varying extent. The purulent exudate in the middle ear cavities showed a distinct fibrin network. There were almost invariably signs of incipient or more advanced organization of the exudate.

4. Krainz, W.: Untersuchungen über die Mastoiditis (Endostitis mastoidea), Ztschr. Hals- Nasen- u. Ohrenh. **13**:361, 1926.

5. Lange, W.: Die pathologische Anatomie der Mastoiditis, Ztschr. Hals- Nasen- u. Ohrenh. **20**:3, 1928.

6. Singer, L.: Über entzündliche Erkrankungen des Mittelohres und der pneumatischen Hohlräume des Schläfenbeines, Ztschr. Hals- Nasen- u. Ohrenh. **32**:130, 1932.

7. Döderlein, W.:: Organizationsvorgänge an den entzündlichen Exsudaten im Mittelohr, Ztschr. Ohrenh. **79**:1, 1920.

8. Wittmaack, K.: Über die normale und die pathologische Pneumatisation des Schläfenbeines: Einschliesslich ihrer Beziehungen zu den Mittelohrerkrankungen, Jena, Germany, Gustav Fischer, 1918.

Thus, the present series seems to support the opinion that only exudate containing fibrin undergoes organization. But fibrin as such hardly is a decisive factor. The process of organization is conditioned by connective tissue proliferation, which is one of the reactions of the organism against damage by inflammation. The presence of exudate containing fibrin generally indicates that these injuries, and mostly also the subsequent proliferative occurrences, exceed a certain limit. As far as the middle ear cavities are concerned, it seems that such an inflammation generally destroys the lining epithelium, at least in some areas. These two factors, i. e., proliferative inflammation and destroyed lining epithelium, evidently are of essential importance in the process of organization. It may be possible that these factors sometimes are active also in middle ear inflammations with afibrinous exudate (Döderlein [7]), but such cases, no doubt, are rare.

From the above observations it may be deduced that tissue injuries exceeding a certain minimum of severity are the first prerequisite for organization of exudate, provided, of course, that the exudate remains in the middle ear cavities for a certain time. This deduction brings up the question: By what route does the exudate leave the middle ear?

The ability of the epithelium to resorb certain substances varies according to its type. The power of resorption of the middle ear epithelium is not definitely known. It seems that it is able to resorb water-soluble substances. Opinions differ regarding the resorption of colloids and solid particles. In the epithelial cells of the middle ear Goerke [9] found pigment particles which were to all appearances derived from the broken-down red blood corpuscles in the exudate. Singer [6] demonstrated by animal experiments that the epithelium of the middle ear is able to resorb particles of India ink, for instance.

The present material included several cases in which epithelium-lined cysts had formed in the middle ear as a result of previous inflammations. Different products of disintegration were always present in these cysts, which, judging from the past histories, must have been formed years before. It seems that the resorptive activity of the middle ear epithelium has but little practical importance as far as the solid particles are concerned.

In addition to the above-mentioned disintegration products, macrophages were a very common finding in these cysts and also in the surrounding connective tissue, a statement made also by Singer. Furthermore, macrophages were fairly often encountered in the exudate in cases of acute otitis in which the most active stage of exudation was apparently over. It seems that they have the function of removing inflammatory products from the middle ear, but they are evidently not effective enough in this respect.

This situation being so, mechanical drainage is the only means by which exudate is removed from the middle ear cavities quickly and effectively. This fact is supported, for instance, by the observation that organization processes are most frequent in the apical mastoid cells, in the small air cells around the antrum, and in the narrow parts of the tympanum, such as the space of Prussak, the window niches, and the vicinity of the ossicular chain, which have the most unfavorable location from the point of view of drainage. As regards the tympanic cavity, it should be

9. Goerke, M.: Die exsudativen und plastischen Vorgänge im Mittelohr, Arch. Ohrenh. **65**:226, 1905.

mentioned, however, that the lining epithelium just in the areas mentioned is most atrophic and endothelium-like. Such an epithelium is easily destroyed during middle ear inflammation because of its low resistance. This condition, no doubt, plays an important part in the distribution of organization processes in the tympanic cavity. On the other hand, all air cells have a similar endothelium-like lining epithelium. The varying frequency of organization processes in different parts of the cellular system can be explained only by different conditions of drainage. It was mentioned above that in some respects otitis media can be compared with pneumonia. According to Mallory,[10] organization of exudate in the pulmonary alveoli is commoner in pneumonia of asthmatic persons, probably also, at least in part, owing to the impeded drainage.

In the middle ear the Eustachian tube is a natural drain which seems to suffice alone, e. g., in sterile foreign body otitis. But in connection with other inflammations of the middle ear the swelling due to inflammation generally blocks the tube more or less completely for a period of varying length. It must be assumed that rapid and complete escape of the exudate is then possible only if pathological channels of drainage are also formed.

Goerke [9] was the first to note that extensive organization processes are common especially in asymptomatic otitis in which the drum membrane does not rupture spontaneously. Otitis of this type is frequently found in persons whose power of resistance is lowered, e. g., by high age or prolonged illness. The majority of the present cases are just of this kind. In altogether 56 temporal bones acute inflammation was observed, in 14 with purulent exudate, whereas in the others the secretion was more serous or mucous. In 3 of the 14 cases the inflammation was severe. Its duration was not definitely known, but it probably cannot have been more than a few days. The tympanic membrane was in the process of being perforated. The middle ear contained much purulent exudate, but there were no signs of beginning organization. Apparently the inflammation had not yet progressed to the proliferative stage. In the remaining 11 cases the drum membrane was to some extent infiltrated with inflammatory cells, but there were no necrotic areas suggesting perforation. The middle ear cavities were filled with fibrinous purulent exudate. At least in two cases in which the otoscopic findings three weeks before death indicated slight middle ear inflammation the histologically demonstrated otitis had probably been of fairly long standing. In all cases the exudate was undergoing organization.

The present series included no cases of acute perforating otitis; thus, no direct comparison of the extent of the organization processes in perforating otitis with that in nonperforating otitis was possible. With the hope of getting some indirect evidence, the tympanic membranes in cases of severe intratympanic adhesions were compared with cases of sparse adhesions or none. The results appear in Table 1.

Data in Table 1, which also includes the 13 cases of clinical adhesive otitis, are based only on histological findings. The intratympanic adhesions were defined as "severe" if there were so many and so firm adhesions round the ossicular chain to the tympanic membrane and in the window niches that they without doubt greatly limited the mobility of these structures. The group "moderate" includes cases in

10. Mallory, T.: Pathology of Pulmonary Fibrosis Including Chronic Pulmonary Sarcoidosis, Radiology **51**:468, 1948.

which adhesions were fairly numerous but were thin and so located that their clinical importance was probably slight. In the third group the adhesions may be regarded as of no clinical significance. Two cases of permanent perforation in the drum were excluded from the table. The "pathological changes" in the tympanic membrane include diffuse or limited cicatricial thickening and limited areas of atrophy. Only those atrophied areas in which the lamina propria of the drum showed a distinct, sharply outlined defect were regarded as "perforation scars."

Table 1 shows that the proportion of severe adhesions is twice as large in cases with no definite signs of previous perforation in the tympanic membrane as in those showing a distinct perforation scar. Figure 1 presents two illustrative cases. As regards the cases of severe adhesions with a perforation scar, it may be assumed that there had been nonperforating otitis previously or that the disease had begun as asymptomatic otitis and the tympanic membrane had ruptured at a later stage. It may be deduced, as far as indirect evidence allows conclusions, that effective drainage of the middle ear sufficient to prevent extensive organization of exudate is possible only if the drum membrane is perforated in good time.

TABLE 1.—*Relationship of Intratympanic Adhesions to Pathological Changes in Tympanic Membrane*

Drum Membrane	Intratympanic Adhesions							
	Severe		Moderate		Mild or Absent		Total	
	No.	%	No.	%	No.	%	No.	%
Normal	0	0	0	0	12	100	12	100
Pathological changes but no perforation scar	13	24	19	35	22	41	54	100
Perforation scars	4	12	11	33	18	55	33	100

But the draining effect of a perforated drum has certain limits. First, in cases with fibrinous exudate the drainage is not possible before the fibrin network has been resolved. Secondly, in cases in which the exudate is not under pressure the drainage is dependent on gravitation. Under these conditions it is evident that the time of the onset, as well as the speed and the extent of the proliferative occurrences in the damaged tissues, must have a great influence upon the extent of the organization processes. Unfortunately, there is very little possibility of evaluating those important factors determined by the individual biological properties of the organism and by the environmental stress, e. g., the species of infectious agent and its virulence.

According to Albrecht [11] and Schwarz,[12] the defence capacity and mode of reaction of the organism to injurious outward agencies are related to the general structural and functional properties of the supporting tissue which together form the body type. Also, the biological properties of the mucous membrane vary with the body type. Thus, for instance, a person of asthenic body type usually has a constitutionally "hypoplastic" mucous membrane, while a person of sthenic type has a "hyperplastic" mucous membrane constitution. The former is liable to alterative,

11. Albrecht, W.: Über Konstitutionsproblem in der Pathogenese der Hals-, Nasen-, und Ohrenheilkunde, Ztschr. Hals- Nasen- u. Ohrenh. **29**:19, 1931.

12. Schwarz, M.: Die Konstitution der Scheimhaut, Ztschr. Hals- Nasen- u. Ohrenh. **40**:332-357, 556-573, 1937.

the latter to productive, inflammation. As for the defence capacity, the mucous membrane may in both types be biologically "superior" or "inferior." It might be supposed that persons of sthenic body type with biologically "inferior" mucous membrane are particularly liable to extensive organization processes. This complicated hypothesis cannot be proved on the basis of the present series.

In addition, it must be taken into account that the reaction of an organism is dependent also on the environmental stress. Extensive organization processes seem to be very common in asymptomatic otitis in persons whose general power of resistance has been lowered, e. g., because of prolonged illness. It may be assumed that the reaction of such an organism to otitis, which is an additional stress, is exceptional. Perhaps the lining epithelium in the middle ear is then easily destroyed,

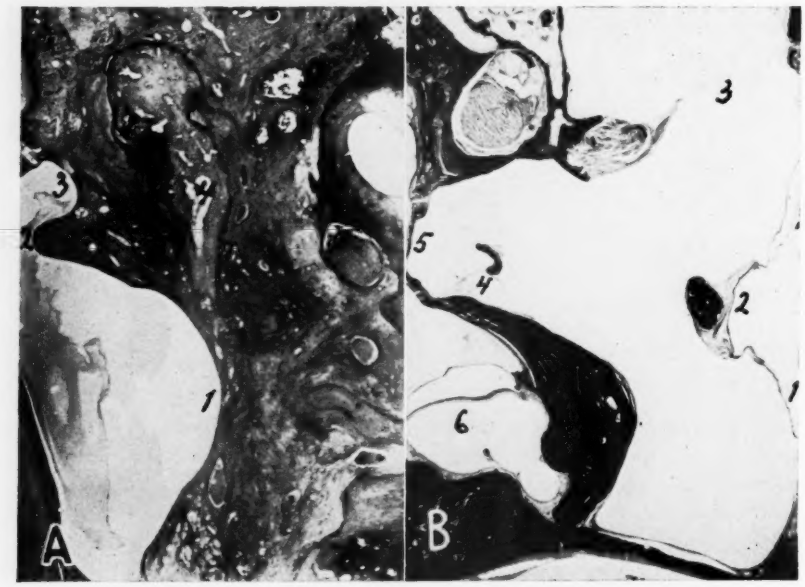

Fig. 1.—*A*, a case in which the past history and the clinical findings suggested adhesive otitis. Otoscopic and microscopic examination did not reveal any perforation scar in the drum membrane (*1*). Processus brevis of the malleus (*2*). Shrapnell's membrane (*3*) forms a deep pouch; incus (*4*) is buried in cystic scar tissue which fills all middle ear cavities. *B*, a case with a large kidney-shaped perforation scar (*1*) around the handle of the malleus (*2*). The mucous membrane of the tympanic cavity is strongly fibrous but fairly thin. Thin adhesion (*3*) from the tendon of the tensor tympani muscle to the lateral wall of the tympanic cavity and from the anterior arm of the stapes (*4*) to the inferior wall of the niche of the oval window. The footplate of the stapes (*5*) and the round window membrane (*6*) are free from adhesions and apparently mobile.

even by fairly mild inflammations. The exudate may, owing to the increased permeability of the capillaries, be more fibrinous than usual, and, perhaps because of insufficient leucocytic reaction, the resolution of fibrin may be disturbed. At least the latter assumption may be partly supported by the observation that a distinct fibrin network was still present in all the present cases in which the exudate was in process of organization. Under these conditions extensive organization of exudate is easy to understand, provided that the organism is still capable of pro-

liferative reaction. From this point of view asymptomatic otitis in connection with other diseases (epidemics, gastrointestinal inflammations of infants, etc.) may be of interest. Experiments with cortisone might also be interesting.

Patients with adhesive otitis often have allergic diseases (Ballenger [2d]). Allergy seems also to play some part in the genesis of postpneumonic pulmonary fibrosis (Mallory [10]). It may be possible that, in addition to the impeded drainage, the special mode of reaction of an allergic organism favors extensive organization processes.

Wittmaack [8] holds the opinion that only the embryonal myxomatous tissue is capable of such an enormous proliferation as is required for extensive organization of exudate in the middle ear. Therefore, according to his opinion, organization processes chiefly occur in early infancy and particularly in connection with "latent" foreign body otitis. Judging from this material, sterile foreign body otitis does not generally appear to cause organization processes. As regards the decisive importance of the persistence of embryonal myxomatous tissue, it should be mentioned that in general pathology such a prerequisite does not exist. Several authors (e. g., Krainz,[4] Lange,[5] Singer,[6] Döderlein,[7] Goerke [9]) have found extensive organization processes in connection with acute otitis at all ages, and this observation is supported by the present series. However, Wittmaack's idea regarding the great practical importance of otitis in infancy must undoubtedly be considered correct. At this early age nonperforating otitis easily escapes notice and when left untreated results in extensive organization of exudate also without any special tendency of the myxomatous tissue to proliferative reaction.

As regards the importance of the infectious agent, it should be mentioned that both the amount of fibrin in the exudate and the time of the onset of proliferative changes in the damaged tissues are partly dependent upon the bacterial strain. Haymann's [13] animal experiments show that in middle ear inflammations caused by Diplococcus pnuemoniae, Type III, connective tissue proliferation leading to organization of exudate may begin during the first three to four days of inflammation. On the other hand, e. g., in diphtheric otitis, proliferative changes take place late, and extensive organization processes seldom occur, in spite of a very fibrinous exudate.

In summary, the theoretical main prerequisites for extensive organization of exudate in the middle ear seem to be (1) that inflammatory damage to the tissues exceeds a certain minimum, being generally indicated by the presence of exudate containing fibrin; (2) that the subsequent proliferation of connective tissue is rapid and powerful, depending upon the individual biological properties of the organism and on the environmental stress, e. g., the virulence and species of the infectious agent; (3) that drainage is impeded, in clinical practice seeming to be owing chiefly to the tympanic membrane rupturing late or not at all.

COURSE OF THE ORGANIZATION PROCESS

The different stages of the organization process have been thoroughly described by Singer,[6] Döderlein,[7] Wittmaack,[8] Goerke,[9] etc., and there is nothing essentially new to be added to their descriptions. Briefly, the process is as follows: The

13. Haymann, L.: Experimentelle Studien zur Pathologie der akutentzündlichen Prozesse im Mittelohr, Arch. Ohrenh. **90**:267, 1913.

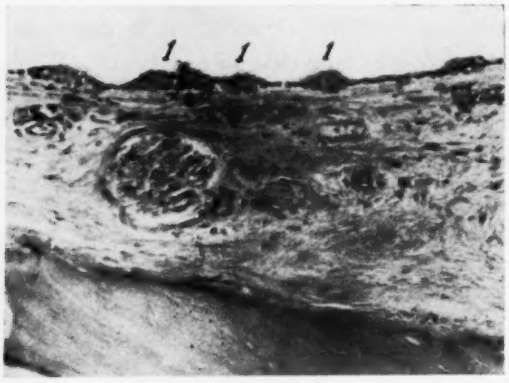

Fig. 2.—The mucous membrane of the tympanic cavity in a case of incipient otitis media. In the submucosa are seen engorged capillaries and a sparse leucocytic infiltration. Small aggregations of pus cells just under the epithelium form slight protrusions (*1*).

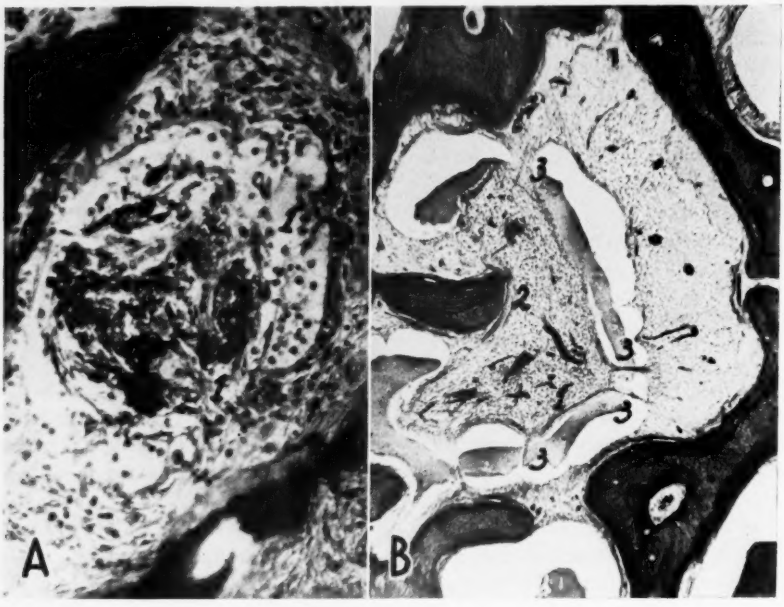

Fig. 3.—*A*, fibrinous exudate in a mastoid air cell in process of organization. Through the previous exudation granulomas (*1*) connective tissue is seen growing into exudate. *B*, a later phase of the organization process. The surface zone (*1*) of the organized tissue which has a broad base (*2*) in one direction is already scarred and has began to contract; the thin connective tissue bridges (*3*) in other directions are stretched; the free lumen is partly filled by newly formed serous exudate.

inflammation seldom extends uniformly throughout the whole submucosa. Usually the superficial layer is most infiltrated with inflammatory cells but in varying degrees in different areas. In some places particularly dense foci form which cause slight protrusions in the epithelium (Fig. 2). At the head of the protrusions an opening forms in the epithelium through which purulent exudate enters the middle ear cavities. The above-cited authors term these formations "exudation granulomas." Exudation takes place also through clefts in the epithelial lining, even when there are no actual openings in the epithelium. When the inflammation has attained the stage of proliferation, the destroyed areas of submucosa are replaced by granulation tissue which grows through the epithelial gaps also in the exudate in the air space. There it spreads fanwise and meets the granulation tissue projecting from the neighboring granulomas. The epithelium of the preserved areas spreads to line this newly formed tissue. Thus, a very characteristic picture arises. In the middle of the cavity is exudate in process of organization. Thin connective tissue columns run from it to the mucous membrane (Fig. 3 *A*). Between the columns there remain cavities lined by epithelium which in the histological section are seen as a string of pearls surrounding the exudate. If the epithelium has been destroyed over a large area, these columns are massive (Fig. 3 *B*), or the exudate becomes organized as in serous cavities so that the boundary of the original mucous membrane can no longer be distinguished. The cavity may in these cases be filled entirely with newly formed connective tissue. The exulate at the periphery becomes organized at first, and the process advances zone by zone towards the center, where there may still be an aggregation of pus cells resembling an abscess when the periphery is already undergoing scar formation.

LATER CHANGES IN THE ORGANIZED TISSUE AND ORIGIN OF ADHESIVE OTITIS

The question seems chiefly to be connected with the postinflammatory repneumatization of the middle ear, viz., the re-formation of the air space lost as a result of inflammation. I have previously [3] dealt in detail with this process, which can be regarded as a pathological variation of physiological pneumatization. A condensed account of the nature and course of physiological pneumatization seems, therefore, to be appropriate. My opinion presented in the study referred to above fundamentally agrees with that earlier presented by Rüedi.[14]

The process of pneumatization is made up of two components differing in basic biological character: preformation of bone cavities and air space formation. The first is not of interest in this connection and will be disregarded here. Air space formation is a phenomenon based on atrophy of disuse and conditioned by certain mechanical factors. During the fetal period the preformed middle ear cavities are for the most part filled with embryonal myxomatous tissue. This serves only as a temporary filling tissue which is replaced with "inert material," with amniotic fluid during the fetal period, with air after birth. The mucous membrane in the middle ear, especially in the air cells, has no mucosal function proper and because of this situation does not differentiate but atrophies until there remain only the lining epithelium and a thin layer of periosteum required for nutrition. This atrophy of living filling tissue presupposes that the resulting "empty space" can be immediately

14. Rüedi, L.: Die Mittelohrraumentwicklung vom 5 Embryonalmonat bis zum 10 Lebensjahr, Acta oto-laryng., Supp. *22*, p. 1, 1937.

and physiologically replaced by "inert material." This occurs through the tube. It does not matter whether the "inert" filling material is amniotic fluid or air. It is only important that the physiological equality of pressure is maintained. The atrophy of living tissue stops if pathological phenomena, for example, transudation and edema *ex vacuo,* are required for equalizing the pressure. To define this briefly, pneumatization of the middle ear is a mesenchymal process conditioned by mechanical factors and caused by biological factors.

Owing to the inflammatory process, there is a change-over from atrophy to proliferation of the living filling tissue; thus, the air space is reduced. When there is no longer any irritation causing tissue proliferation, the air space again begins to enlarge if the mechanical prerequisites are present. The process differs from the physiological pneumatization in that the structure and properties of the living filling tissue are pathologically altered. This alteration depends upon the intensity and especially on the duration of the inflammation.

If the inflammation subsides rapidly, the connective tissue proliferation will also rapidly cease, as there is no longer fresh tissue damage. The young granulation tissue remains poor in cells and fibers and resembles, in structure and apparently also in its biological properties, embryonal connective tissue. If the mechanical prerequisites are present, viz., aeration through the tube is free, this tissue seems to be able to atrophy almost to the same extent as embryonal connective tissue. The proliferated submucosa, therefore, again becomes "mucoperiosteal," though the number of cells and fibers increases in varying degree. Owing to the structure of the granulation tissue and its localization mainly to circumscribed foci, mucosal fibrosis in these cases seems generally to remain slight, from which observation it follows that physiological pneumatization proceeds without disturbance after repneumatization is complete.

The "balls" of organized tissue in the middle ear cavities also contract and atrophy. My series shows different stages of this development (Figs. 2, 3, and 4). The tissue "balls" contract concentrically. The connective tissue bridges uniting them with the mucous membrane stretch correspondingly. As the organization of exudate takes place gradually and advances towards the center, the newly formed tissue also contracts zone by zone. In this way characteristic lamellated nodules resembling onions arise. Photomicrographs of these formations were presented in my previous study.[3] The vascular connective tissue bridges stretch farther and become thinner and thinner, and the nutrition diminishes continuously. The end-result of this process is that the fibrocytes die and the nodule apparently undergoes calcification and, after all connecting bridges have broken, remains isolated in the cavity, resembling to some extent a corpus amylaceum (Fig. 4 *A*). If the "ball" of organized tissue is in any direction connected with the mucous membrane by a massive connective tissue base (Fig. 3 *B*), it contracts chiefly in that direction, while the connecting bridges in other directions stretch and break off. The "nodule" is then better nourished. Therefore, the epithelium and the superficial fibrocytes may survive (Fig. 4 *B*), and dystrophic calcium deposits occur only in the center. In some cases new bone formation may be encountered (Fig. 5). German [15] and

15. German, T.: Uber die Entstehung multipler Knocheneinschlüsse im Warzenforsatze, Ztschr. Hals- Nasen- u. Ohrenh. **18**:339, 1927.

Horowitz [16] have also reported cases in which probably the organized tissue in the tympanic cavity or in mastoid air cells has become ossified. Such a development seems to be rare. On the other hand, onion-like nodules of the kind described above are fairly common. They were found in 19 of the 101 adult specimens in the present series, or in 19%. Kessel [17] gives a description and drawing of apparently the same formations but considers them physiological "organs" having the function of perceiving the changes in pressure and tension conditions in the middle ear. He states that Politzer has expressed the same opinion. Goerke [9] says that he has found corpora amylacea in the mastoid air cells. In these cases, too, there have probably been residua of organized tissue like those described above.

In some cases of the present series a fine network of connective tissue fibers apparently represented the final result of organized tissue. All these residua prob-

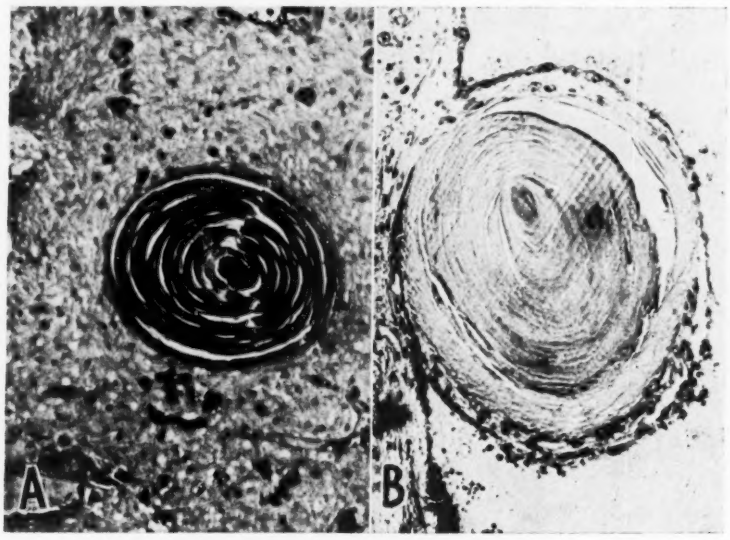

Fig. 4.—*A*, an isolated calcified nodule representing the final result of organized exudate when repneumatization has been completed and all connecting bridges have broken. The nodule has been buried in newly formed organization tissue in the course of a later middle ear inflammation. *B*, the final result in a case in which the organized tissue has had a broad base in one direction. The connecting bridges in the other directions have broken, the center of the nodule is acellular and calcified, but the lining epithelium and the superficial fibrocytes have survived.

ably lack clinical significance. The well-known fact that acute otitis of short duration seldom causes residua which affect the hearing can thus be histologically explained. It should be noted that the mechanical prerequisites for repneumatization must then be present.

16. Horowitz, S.: Heterotopic Ossification in the Middle Ear, J. Laryng. & Otol. **66**:181, 1952.

17. Kessel, J.: Die Histologie der Ohrmuschel, des äusseren Gehörganges, Trommelfells, und Mittelohrs, in Schwartze, H.: Handbuch der Ohrenheilkunde, Leipzig, F. C. W. Vogel, 1892, Vol. 1, pp. 43-101.

If the original inflammation is protracted, changes will arise in the structure of the organized tissue which affect the repneumatization decisively. The inflammatory reactions of the periosteum and bone tissue in general, i. e., osteoblastic and osteoclastic processes, are of less importance in connection with adhesive otitis and will be disregarded here.

Owing to a prolonged inflammation, there is always fresh tissue damage and tissue proliferation. As a result of this, the number of cells and fibers in the granulation tissue constantly increases. This increase does not occur evenly throughout the whole submucosa but chiefly in the most severely affected surface zone adjoining the lumen. The result may be that after the inflammation finally has subsided, an unyielding cicatricial wall has formed under the epithelium (Fig. 6*A*), and it encloses the deeper tissues adjoining the bone walls within an inelastic frame on all

Fig. 5.—Bone formation (*1*) in the organized tissue in the lateral part of the epitympanum. Head of the malleus (*2*); the lateral wall of the attic (*3*); the lateral malleal ligament (*4*).

sides. The tissue thus encapsulated is, in a way, in the position of bone marrow. It cannot be physiologically replaced by "inert material," and therefore it retains its function of filling tissue. Thus, the loss of the air space is no longer reversible. On the contrary, as long as there is active inflammation causing fresh exudation, the reduction of the free lumen continues. The "pearl string" cavities surrounding the original "ball" of organized tissue fill with new exudate (Fig. 8) which becomes organized. The process repeats itself, and finally the free lumen may be entirely destroyed, or parts of it are isolated and transformed into epithelium-lined cysts (Fig. 1*A*). The organism is not able to eliminate the contents of these cysts which then, together with the unyielding cicatricial walls described above, constitute a permanent hindrance to repneumatization. Naturally, physiological pneumatization is then also at an end.

It should be mentioned that even middle ear inflammations of short duration may lead to the same result if there are frequent relapses and the attacks follow each other before repneumatization has had time to take place.

The later development depends upon the inflammatory process. If it subsides altogether, the dense scar tissue contracts until the traction components acting in different directions have reached a position of equilibrium. The looser connective tissue "encapsulated" between the inelastic cicatricial walls and the bony walls can no longer contract and atrophy. It persists as a filling tissue. Often it seems, like bone marrow, to be transformed into fatty tissue (Fig. 6*A*).

In some cases this tissue persists without undergoing any changes (Fig. 6*B*). This condition seems to be due to slight inflammatory irritation which causes no

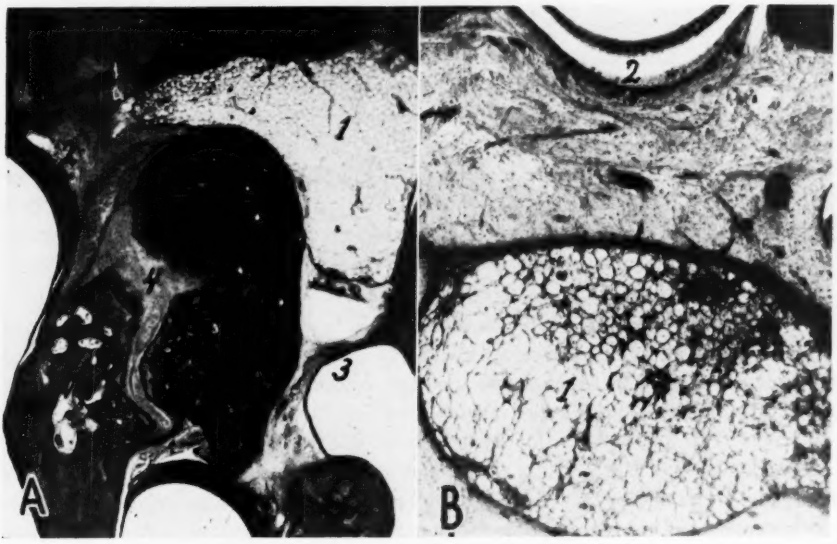

Fig. 6.—*A*, organized tissue enclosed in the lateral part of the epitympanum (*1*) has been transformed into fatty tissue; it is separated from the free epitympanum by an inelastic wall of scar tissue (*2*); Prussak's space has been obliterated, and Shrapnell's membrane has been drawn inward to the lateral malleal ligament (*3*); the incudomalleal joint (*4*). *B*, a case in which a simple mastoidectomy had been performed two years previously. After this there had been no symptoms of inflammation in this ear; the air cells which had not been evacuated at operation are filled with organized tissue containing great cyst cavities (*1*) here and there; a thin fibrous zone (*2*) separates the organized tissue from the free lumen; otherwise, the organized tissue probably formed two years ago has remained loose and edematous.

tissue destruction but maintains a "chronic phlegmasia." Apparently the cysts containing unabsorbed disintegration products constitute such an irritating factor.

During prolonged or frequently recurring inflammation the organized tissue in the middle ear cavities is also transformed into increasingly dense cicatricial tissue which is able to contract to a certain point but has only a slight tendency to atrophy. In this way permanent firm cicatricial cords and folds form which are fairly well nourished and therefore, contrary to the onion-like nodules described above, seldom contain abundant calcium deposits. If the inflammation definitely ceases after this,

these pathological formations become sites of regressive changes, but it is probable that clinically the situation in the main seems to be stationary; i. e., there are otitic residua with adhesions.

On the other hand, if there are continually frequent relapses, it is probable that a progressive deafness having the well-known clinical characteristics of chronic adhesive otitis develops. The adhesive folds and cords render drainage poor; therefore every new attack ends in the formation of new adhesions and increasing hearing loss. Latent foci of infection may be retained in the narrow-mouthed blind pouches formed by the adhesions and in the cysts, which are very common, particularly in the mastoid air cells. A local predisposition to recurring acute exacerbations is then present. In addition, there must also be other predisposing factors, both congenital, such as inferior resistance and exceptional reaction to outward agencies, e. g., allergy, and acquired, e. g., chronic inflammatory, processes in the nose, the nasal accessory sinuses, and the nasopharynx and different pathological conditions leading to Eustachian tube obstruction. A certain familial tendency to adhesive otitis (Lumio [1]) can be explained only on the basis of congenital factors. The fact that adhesive otitis is usually bilateral (for example, in 181 of Lumio's 185 cases) also argues in favor of congenital factors. In some cases syphilis may be of importance in the etiology (Saxén,[18] Saxén and Ojala [19]). In two of the cases of severe chronic adhesive otitis in the present material there was tertiary syphilis, and in a third case the autopsy findings and histological examination clearly indicated syphilis, though this could not be confirmed, as serological tests had not been made. Tuberculous otitis may also be an etiological factor of some importance.

Wittmaack [2c] considers that adhesive otitis is based on regressive changes in the pathologically altered submucosa. Owing to the poor nutrition of the cicatrized mucous membrane, these dystrophic changes continue, although there is no longer any inflammation. True adhesions may be wholly absent. During this process dystrophic calcium deposits or newly formed bone are laid down in the submucosa, in the cartilages of the ossicular joints, and in the ligaments. A bony fixation of the footplate of the stapes may then develop having no relationship to otosclerosis. In such a case there is a clinical picture of chronic adhesive otitis but without adhesions. Because adhesions do not seem to be necessary, Wittmaack proposes that this disease be termed a "regressive process."

The present cases included none showing a "regressive process" of this kind. It is true that in the cases in which the conditions for repneumatization evidently had been favorable the organized tissue in the middle ear cavities was transformed, owing to regressive changes, into acellular, probably calcified nodules. However, there were no signs of dystrophic calcium deposits in the original mucous membrane, although it was much scarred. Such changes seemed to be fairly rare also in cases in which the scarred adhesive bands were thick and had broad bases and because of this were well nourished. As far as conclusions can be drawn from decalcified

18. Saxén, A.: Pathologische Anatomie und Klinik der degenerativen Erkrankungen des Gehörorgans, Ergebn. allg. Path. u. path. Anat. (Supp.) **34**:134-157, 1938.

19. Saxén, A., and Ojala, L.: Über spätluetische und durch latente Otitis verursachte Veränderungen im Gehörorgan: Diskussion auf Grund eines Falles, Arch. Ohren- Nasen- u. Kehlkopfh. **160**:11, 1951.

material, I am inclined to believe that dystrophic calcium deposits are of minor importance in the pathology of chronic adhesive otitis. Probably regressive changes generally result in a clinically stationary condition, or, in other words, in otitic residua. No doubt, in most cases the disease is clinically progressive only if there

Fig. 7.—The head of the malleus (*1*) is fixed to the roof of the epitympanum (*2*) by firm cicatricial tissue and newly formed bone.

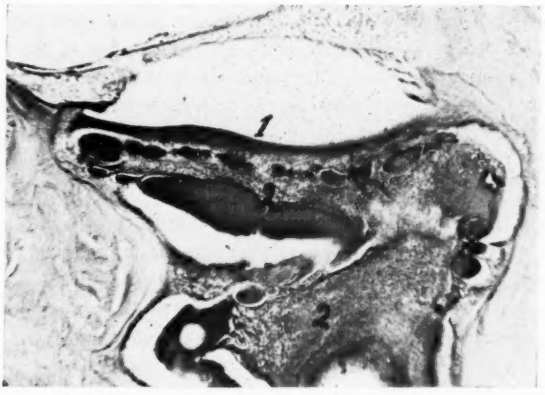

Fig. 8.—Organized tissue in the niche of the round window. The "pearl string" lumen between the round window membrane (*1*) and the organized tissue (*2*) is filled by newly formed serous exudate.

is a continuous or recurring inflammation causing connective tissue proliferation. The cases of bony fixation of the stapes without adhesions described by Wittmaack may belong to another category, perhaps to those diseases of unknown origin that lead to metaplastic bone formation in the mucous membrane, e. g., tracheopathia

osteoplastica. In some cases bony fixation of the ossicular chain is apparently due to inflammatory new bone formation. Among the present cases there was one of this kind in which the malleus was fixed to the roof of the tympanic cavity by firm adhesions and newly formed bone (Fig. 7). But in the tympanic cavity the inflammatory new bone formation generally seems to be slight, although in the mastoid air cells it may be very abundant. In true adhesive otitis connective tissue adhesions are, no doubt, the main cause of the immobility of the middle ear structures.

Thus, the classical designation, chronic adhesive otitis, seems well to characterize the disease. But there is scarcely reason for a strict classification of the cases into two groups, otitic residua with adhesions and adhesive otitis. Progressive adhesive otitis may change into clinically stationary otitic residua if inflammations no longer occur, and vice versa, stationary otitic residua with adhesions may, owing to recurring inflammations, change into progressive adhesive otitis.

COMMONEST SITES OF ADHESIONS AND THEIR CLINICAL IMPORTANCE

Naturally, the adhesions are most extensive at the sites of predilection of the organization processes. In the tympanic cavity these are, in order of importance, the window niches, Prussak's space, and the epitympanum, especially its lateral part.

In all cases of severe adhesions there were adhesions in the niche of the oval window. They chiefly stretched from the arms of the stapes to the walls of the niche, most and the thickest of the adhesions from the anterior arm of the stapes to the anterior and superior walls of the niche. As is known, the footplate of the stapes moves like a window which "opens" anterosuperiorly and has its "hinges" posteroinferiorly. The nearer the anterior edge the adhesions are, the more effectively they thus "lock" the window. The interesting frequency of this site in the present cases may be due to chance, but certain anatomical circumstances support the view that this region may be particularly liable to adhesions. The membrana obturans stapedis prevents the escape of exudate from the upper part of the niche between the arms of the stapes. The processus cochleariformis and the tendon of the tensor tympani muscle form the boundary of the anterosuperior part of the niche. Around both of these structures exudate very often seems to become organized, and drainage in the anterior direction is therefore obstructed. Under these conditions organization of the exudate may be more extensive in the anterosuperior part than in other parts of the niche.

In all cases of severe adhesions there were adhesions also in the niche of the round window. In two cases they were limited to the lateral part of the niche; in the others a varying number of adhesions were attached to the window membrane, evidently limiting its mobility (Fig. 8). The present cases seem to show that in adhesive otitis pure ankylosis of the stapes is rare; mostly the round window membrane is also fixed to a varying extent. It is worth noticing that adhesions to the round window membrane were not infrequent, even in cases with very sparse intratympanic adhesions. This observation may be of some clinical interest because the mobility of the round window membrane may be limited even by adhesions which are so thin that they would scarcely have any importance elsewhere in the tympanic cavity.

In all the present cases of severe intratympanic adhesions Prussak's space contained varying amounts of fibrous cicatricial tissue. This caused deep indrawing

of Shrapnell's membrane in 12 cases. In five cases of moderate adhesions Shrapnell's membrane also showed a deep depression due to adhesions. On the other hand, indrawing of Shrapnell's membrane was never observed in the cases of sparse intratympanic adhesions or none. From this finding it may be concluded that a depression in Shrapnell's membrane which does not disappear after successful inflation of the middle ear and is thus due to adhesions proves definitely that there are adhesions also elsewhere in the tympanic cavity. In doubtful cases this may be helpful in differential diagnosis.

In the epitympanum adhesions from the malleus to the lateral wall and to the roof of the tympanic cavity were commonest. In the cases of severe adhesions they were so extensive and firm that the malleus would evidently have appeared fixed on clinical examination (Fig. 7). Adhesions starting from the incus were rarer. In cases of severe adhesions the incus in eight cases adhered to the medial wall of the tympanic cavity. In six cases there was a firm adhesion from the tip of the long process of the incus to the tympanic membrane. In five cases of moderate adhesions the long process of the incus was also adherent to the drum. Adhesions between the drum membrane and the promontory were infrequent. They were found only in three instances.

Unfortunately, in the present series a reliable comparison of the results of functional tests of hearing with the extent and location of the intratympanic adhesions, as well as a histological study of the effect of adhesive otitis upon the inner ear structures, was not possible because of degenerative inner ear changes due to the high average age of the patients. Nevertheless, I should like to present some hypotheses regarding these interesting questions.

Involvement of the inner ear seems to be common, even at early stages of adhesive otitis (Lumio[1]). For the most part this may be due to toxic serous labyrinthitis causing degenerative changes in the inner ear structures (Wittmaack,[2c] Saxén,[18] Kopetzky,[20] and others). Several facts support this assumption. Adhesions to the round window membrane were frequent, even in cases of fairly sparse intratympanic adhesions, indicating that there must have been inflammatory foci in this thin membrane. Toxic products of bacterial activity may then have easily diffused into the perilymph. During acute exacerbations the "pearl string" cavities between adhesions fill with exudate (Fig. 8). Because of the poor drainage absorption of toxins into the perilymph may then be more extensive than in simple acute otitis. Owing to recurring inflammations, some of the "pearl string" cavities may be transformed into cysts containing toxic products of disintegration. It is easy to understand that toxic labyrinthitis of long duration may then follow. It is also possible that the contents of the cysts, which are very frequently found especially in the mastoid air cells, act as a "focus" producing allergens. Allergic labyrinthine hydrops may then develop, with its deleterious effect upon the inner ear structures.

Serous labyrinthitis is not the only explanation of the perceptive deafness observed in connection with adhesive otitis. As is known, fixation of the round window membrane may result in deafness of inner ear type. Individual adhesions to the round window membrane were not infrequently found, even in cases in which there was a lack of other significant adhesions in the tympanic cavity. A loss of

20. Kopetzky, S.: Deafness, Tinnitus, and Vertigo, New York, Thos. Nelson & Sons, 1948.

hearing of inner ear type following acute otitis (Poulsen [21]) or at the early stage of adhesive otitis (Lumio [1]) may be caused partly by limitation of the mobility of the round window membrane due to such adhesions. According to Krainz,[22] this "window membrane deafness" can be distinguished from the true nerve deafness by means of Gellé's test as modified by him.

Tinnitus and vertigo accompanying head colds apparently have their explanation in serous labyrinthitis. On the other hand, vertigo in connection with inflation of the middle ear may be chiefly due to adhesions between the incudostapedial joint and the tympanic membrane. Such adhesions are not infrequent. In the present material the tip of the long process of the incus adhered to the tympanic membrane in 11 cases in all, or in 23% of the total number of the cases of severe and moderate intratympanic adhesions (47 cases). Normally very great movements of the tympanic membrane result in dislocation of the incudomalleal joint. The articular surfaces glide over one another, thus reducing the amplitude and force of the vibrations and protecting the inner ear structures from too severe irritation (Kopetzky [20]). This protecting mechanism is out of action if the tip of the long process of the incus adheres to the drum. The bulging of the drum membrane due to inflation of the middle ear may then result in irritation of the static end-organs, naturally provided that the mobility of the footplate of the stapes is not strongly limited. Thus, vertigo in connection with inflation of the middle ear in cases of adhesive otitis may be a sign indicating that the conductive type of deafness is caused by immobility of other parts of the conductive mechanism and not by ankylosis of the footplate of the stapes. From the point of view of fenestration this may be of practical importance; in such cases favorable results cannot be expected.

MASTOID PNEUMATIZATION IN CONNECTION WITH ADHESIVE OTITIS

Because of the small number of cases of clinical adhesive otitis in the present series and because of the fact that histopathological changes affecting pneumatization must have been essentially the same although of different degree both in cases resulting in stationary intratympanic adhesions and in those leading to progressive adhesive otitis all cases of intratympanic adhesions were considered in studying mastoid pneumatization. The material is, even then, numerically too small and, in addition, too inhomogeneous for statistics, but some histological observations and theoretical conclusions may be of interest.

In cases of severe and moderate intratympanic adhesions the mastoid air cell system almost invariably showed different sequelae of asymptomatic mastoiditis (Fig. 9*A*). In five cases of severe adhesions the whole air cell system, including the antrum, had been totally destroyed and transformed into groups of cystic cavities. In nine cases these changes were less extensive, but a large number of the air cells had been filled with fibrous tissue or had changed into cysts. The mucous membrane in the antrum and in the remaining air cells was rather thick and scarred. Across the lumen there were often adhesive bands and cords, or large "lumps" of

21. Poulsen, I. P. V.: Otitis Media Acuta in Children 0-3 years of Age, Acta oto-laryng. **38**:120, 1950.

22. Krainz, W.: Die funktionelle Bedeutung des runden Fensters, Ztschr. Hals- Nasen- u. Ohrenh. **18**:433, 1927.

cicatrized organized tissue, and mucous or serous secretion with sparse lymphocytes and macrophages. In three cases the mucous membrane was fairly thin but strongly fibrous, and across the lumen there were abundant thin adhesive cords. Around the air cells there were cavities filled with fibrous connective tissue. In some of these careful study revealed individual isolated acellular nodules (Fig. 9*B*) buried in the fibrous tissue. Judging from this observation, the cavities were not fibrous marrow spaces but previous air cells. Thus, severe intratympanic adhesions were always accompanied by impeded repneumatization. In all cases the bone structure showed secondary sclerosis of varying extent both in the marrow spaces and in the air cells, especially in those filled with fibrous tissue. In the cases of moderate adhesions the changes described above were of minor degree. Acellular residua of organized tissue

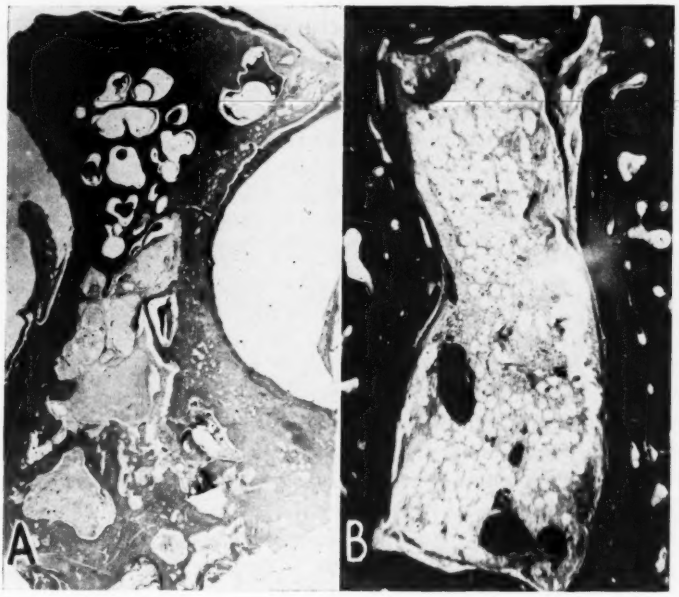

Fig. 9.—*A*, mastoid air cell system in a case of adhesive otitis. The large cavities formed by destruction of the cellular walls are partly filled with organized tissue and newly formed bone. Marked secondary sclerosis in the neighboring marrow spaces. *B*, acellular residua of organized tissue in a previous air cell. Owing to a later middle ear inflammation, the air cell has been totally filled with new organized tissue; after this repneumatization has no more been possible, and subsequently the latter tissue has been partly transformed into fatty tissue; the fibrous zone in the periphery of the cavity apparently represents the original mucous membrane.

were fairly common, indicating that the conditions for repneumatization had been fairly good. In the cases of mild adhesions complete repneumatization was almost the rule. The mucous membrane was thin but had undergone fibrosis of varying degree, and a few adhesions traversed the lumen here and there.

The extent of mastoid pneumatization in the different groups of intratympanic adhesions appears in Table 2.

In judging the extent of mastoid pneumatization an attempt was made to follow Schwarz's [23] classification of five pneumatization groups (p. g.) Table 2 shows that 53% of the temporal bones in severe intratympanic adhesions were scantily (p. g. IV) or not (p. g. V) pneumatized. As was mentioned above, there was impeded repneumatization in all cases; in the five cases belonging to group V even the antrum was totally filled with cystic organized tissue. The mastoid bone showed secondary sclerosis of varying extent. As regards the cases of moderate adhesions, 67% were well (p. g. I and II) or fairly well (p. g. III) pneumatized. In cases of sparse adhesions or none this percentage was higher still (80%). Thus, it seems that the quality and the quantity of the adhesions have some relationship to the extent of the air cell system. It may be worth mentioning that in the nine cases of severe adhesions belonging to groups IV and V the patients in question could not remember any middle ear inflammation. This fact may be due to the high age of the patients. But it may also be possible that the inflammations had chiefly occurred in childhood or even in early infancy and therefore the patients could not remember them. Naturally, this assumption cannot be proved, but, to some extent, it may support Wittmaack's opinion regarding the causality of otitis in infancy, certain permanent changes in the structure of the tissues of the middle ear, and poor pneumatization. The words "to some extent" mean that in cases in which the reaction of an organism to continuous or frequently recurring environmental stress of certain quality and quantity results in certain permanent structural changes in the tissues of the middle ear already in infancy the pneumatization always remains poor because the growth of the air cell system is no longer possible in every case. It is evident that otitis in infancy does not always result in such permanent structural changes. This is the case only if an environmental stress of a certain character strikes an organism with certain inherited characteristics. It may be possible, as stressed by Wittmaack, that adhesive otitis often starts in infancy. However, the high proportion of cases of severe intratympanic adhesions with good or fairly good pneumatization shows that, at least in the present series, in many cases the process cannot have begun in infancy but later in life.

TABLE 2.—*Mastoid Pneumatization in Different Groups of Intratympanic Adhesions*

	Extent of Pneumatization According to Schwarz's Classification											
	I		II		III		IV		V		Total	
Intratympanic Adhesions	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Severe	..	..	1	6	7	41	4	23	5	30	17	100
Moderate	1	3	6	20	13	44	9	30	1	3	30	100
Mild or absent	3	5	27	52	12	23	5	10	5	10	52	100

A study was also made to determine how often the adhesions were qualitatively and quantitatively nearly similar in both ears of the same person. The 17 temporal bones with severe intratympanic adhesions represented 12 patients, and the 30 temporal bones with moderate adhesions, 26 patients. Thus, the process was bilaterally nearly similar in 41% of the cases of severe adhesions and only in 15% of the cases of moderate adhesions. It should be added that also in cases in which the process

23. Schwarz, M.: Die Bedeutung der hereditären Anlage für die Pneumatisation der Warzenfortsätze und der Nasennebenhöhlen, Arch. Ohren- Nasen- u. Kehlkopfh. **123**:161, 1929.

was not similar in both ears the other ear was never healthy. As regards the cases with severe adhesions in the one ear, in the other ear adhesions defined as moderate were found in four and mild adhesions in three cases. In 18 of the cases of moderate adhesions in one ear there were mild adhesions in the other. As regards the extent of pneumatization, it is worth mentioning that both temporal bones were poorly or not at all pneumatized in four of the five cases of severe bilateral adhesions. All these observations support the view that inherited biological properties of an organism are of great importance in the genesis of adhesive otitis.

Finally, attention should be paid to the fact that severe intratympanic adhesions were almost invariably, and even moderate adhesions often, associated with distinct sequelae of healed mastoiditis. This observation is in accordance with the view that permanent adhesions mostly develop in connection with a prolonged inflammation. Previously, however, many cases of purulent acute mastoiditis if operation was not performed resulted in chronic mastoiditis and chronic manifest suppurative otitis instead of spontaneous cure. Nowadays conservative treatment by means of the new drugs may be substituted for surgical treatment in many cases of surgical mastoiditis. But the more prolonged the treatment, the greater is the risk of permanent adhesions and impairment of hearing. In addition, although there is no more manifest suppuration, there may be clinically asymptomatic mastoiditis. If the patient is not under observation, chronic asymptomatic mastoiditis may develop, perhaps leading to chronic adhesive otitis instead of chronic manifest suppurative otitis. For this reason the assumption was presented at the beginning of this paper that some conditions which previously would have resulted in chronic manifest suppurative otitis may now develop into chronic adhesive otitis.

PRACTICAL CONCLUSIONS

The prophylactic treatment of adhesive otitis should fulfil two main conditions: 1. Organization of the exudate in the tympanic cavity must be reduced to a minimum. 2. Unhindered repneumatization must be ensured. A problem of the present day is how to use the valuable new antibacterial drugs most effectively for these purposes.

The recent literature contains statements to the effect that the modern drugs make paracentesis unnecessary (Monteiro [24]). In theory organization processes can be prevented with medication alone if the treatment can be started and the inflammation overcome at so early a stage that no appreciable tissue damage has time to arise. If prerequisites for the organization process are already present at the beginning of treatment, organization of exudate cannot be prevented by means of the antibacterial drugs alone. On the basis of clinical examination there is but little possibility of drawing reliable conclusions from the extent of the inflammatory damage to the tissues in a case of nonperforating otitis. The type of secretion, serous, mucous, or purulent, may be the most useful clinical sign, but it requires myringotomy. The proper chemotherapy presupposes determination of the bacterial strain and its resistance. In nonperforating otitis myringotomy is then necessary. Indiscriminate chemotherapy alone may result only in delayed spontaneous rupture of

24. Monteiro, A.: Sulfas e penicilina no tratamento das otitis médias aguadas e suas complicacões: Estudo comparativo, Hospital, Rio de Janiero **36**:23, 1949; abstracted, Excerpta Med. XI, **3**:28, 1950.

the drum or in the otitis continuing as asymptomatic, with extensive organization of exudate. Such a therapy may provoke, instead of prevent, permanent adhesions and perhaps also development of progressive adhesive otitis. So far as Haymann's [18] results apply to man, organization of exudate may begin during the first few days of inflammation. To prevent this, the exudate must be evacuated from the middle ear as soon and as completely as possible. For all these reasons, in addition to chemotherapy, early myringotomy is to be considered indispensable. But myringotomy as such is no guarantee for complete drainage. The patient must be kept under surveillance, and the escape of the exudate must be ensured, e. g., by inflation of the middle ear, by suction by means of Siegle's speculum, or by puncture of the drum and aspiration by means of needle and syringe. The procedure must be repeated when needed. If the chemotherapeutical agent is properly chosen and administered in sufficient doses, the exudation in most cases rapidly ceases, and the middle ear will remain dry. Thereby the danger of extensive organization of exudate is reduced to a minimum.

The measures for ensuring repneumatization are self-evident, and I shall only make a few remarks upon this question. The ultimate result is most advantageous if the inflammation subsides rapidly and the patient can be safeguarded against recurrences at least until the hearing has returned to normal. Then repneumatization can be considered essentially complete. It is therefore important to continue treatment long enough and to keep the patient under observation until normal hearing is restored. Naturally, any etiological factors causing blockage of the tube and recurrence of otitis must be carefully eliminated. Special attention must be paid to the possibility of asymptomatic mastoiditis. Poulsen [21] is, no doubt, right in postulating that from the point of view of functional results early mastoidectomy is a better solution than prolonged treatment with antibiotics, although the inflammation may finally be cured also by means of the latter method alone. It is evident that difficulties are greatest when the patient is a child. Middle ear inflammations in general are commonest during the first decade of life. These facts support the view that middle ear inflammations of infancy and childhood may be the most important battle front in combating adhesive otitis.

Finally, I wish to express some theoretical points of view as regards fenestration in cases of adhesive otitis. The first prerequisite for favorable results, naturally, is that true ankylosis of the stapes exists. Vertigo in connection with inflation of the middle ear or in connection with a strong negative pressure produced in the external auditory canal, e. g., by means of Siegle's speculum, may speak in favor of mobile stapes. Because the round window after operation is still exposed to adhesions, the prognosis may be more reliable in cases in which the disease has long remained stationary. Therefore, the history should be carefully considered. Prior to operation the etiological factors predisposing to relapsing middle ear inflammations should be eliminated as far as possible.

SUMMARY

On the basis of a histological study of material comprising 116 temporal bones the following conclusions regarding organization of exudate and pathogenesis of adhesive otitis are made:

The theoretical prerequisites for extensive organization of exudate in the middle ear seem to be (1) that the inflammatory damage to the tissues exceeds a certain minimum, being generally indicated by the presence of exudate containing fibrin; (2) that the subsequent proliferative reaction of the connective tissue is rapid and powerful, depending on the inherited biological properties of the organism and on the quality and quantity of the environmental stress, e. g., on the species of the infectious agent and its virulence; (3) that drainage is impeded, in clinical practice probably chiefly owing to the drum membrane rupturing late or not at all.

If the inflammation subsides rapidly and the conditions are favorable for repneumatization, the organized tissue usually atrophies and contracts without causing residua affecting the function of hearing. If the inflammation is protracted or recurs frequently, the organized tissue in the middle ear cavities becomes denser and is transformed into permanent adhesions. After the inflammation subsides, they become sites of regressive changes which finally lead to a clinically stationary condition; i. e., there are otitic residua with adhesions. The disease progresses if recurring inflammations cause recurring tissue proliferation and new adhesions.

The histological characteristics, etiological factors, and therapeutical aspects of adhesive otitis are discussed.

ILLNESS AND DEATH OF AFRANIUS BURRUS

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IN THE time of the ancient Roman emperors cancer was a disease well known not only to members of the medical profession but also to the general public. In his great poem "Metamorphoses" P. Ovidius Naso (43 B. C.—17 or 18 A. D.), a contemporary of Augustus, in relating the petrification of the jealous Aglauros mentions cancer:

"As an incurable evil, cancer usually spreads, joining healthy parts to the sick." [1] This poem was not intended for a selected class of intellectuals but for the more enlightened general populace. It must have been presumed by the poet who penned these lines that the intelligent masses knew about cancer and the most important symptoms of the disease.

Nevertheless, many a case must have passed undetected. Cancer of the internal organs was inaccessible to the diagnostic art of the physicians, and only cancer of the skin or of the mammary gland was known.

Just such an undetected laryngeal carcinoma seems to me to have been the disease of Afranius Burrus, military preceptor of Nero (54-68 A. D.). Historians affirm that Burrus was poisoned by Nero, and these affirmations are based on the writings of Tacitus and Suetonius, both authors very unfavorable to the emperor. By reading the writings of these ancient Roman historians from a medical point of view, without any prejudice against the emperor, one must find that there are very few indications of poisoning and far more indications of natural disease, especially laryngeal carcinoma.

In the year 54 A. D. Agrippina had chosen Afranius Burrus as the military preceptor of her son, wishing to prepare her son to be emperor. The choice was good. Tacitus says of Burrus: "The leadership of the cohorts was conferred upon Afranius Burrus, a man with an excellent war reputation." [2]

Soon thereafter, Tacitus tells us that Burrus was the right man in this position:

Murder was imminent [and would have occurred] if Afranius Burrus and Annaeus Seneca had not presented an obstacle to it. They were the tutors of the young emperor, living in harmony with each other, which is rarely the case with associates in power; and each possessed an equal influence, prevailing over him in different ways: Burrus by his assiduous attention to military affairs and strictness of moral discipline; Seneca by his lessons in eloquence and by his discreet conduct; each assisting the other that they might more easily restrain the prince, at his perilous time of life, within the limits of unforbidden pleasures, if tempted by immorality, he should shun virtue.[3]

1. P. Ovidius Naso: Metamorphoses II, 825-826.
2. P. Cornelius Tacitus: Annales XII, 42.
3. P. Cornelius Tacitus: Annales XIII, 2.

The death of Burrus occurred in the year 62 A. D. Suetonius, in his biography of the emperors,[4] affirms that Nero had sent to his preceptor a poison instead of a remedy for sore throat. For this affirmation Suetonius gives no proof. On the contrary, Arthur Weigall,[5] more favorably disposed toward Nero, affirms that the death of Burrus occurred from a natural disease.

P. Cornelius Tacitus has given a detailed description of the disease and of the death of Burrus:

> But while the distress of the state was every day more oppressive, means of redress during this period diminished. It was at this time that Burrus departed this life, it is not known whether by disease or by poison: disease was inferred from the fact that, his throat gradually swelling internally and the passage being obstructed, he ceased to breathe. Many people asserted that at the command of Nero his palate was painted with a poisonous medicament as if a treatment were undertaken; and that Burrus, who had perceived the villany, when the prince came to visit him, turned his face away from the prince and would reply to his questioning nothing more than, "I am well." A great affection for him remained throughout the nation, remembering his virtue in contrast to the spiritless simplicity of one of his successors, and the most flagrant debauchments of the other.[6]

The medicament, supposed later to be a poison, was evidently not applied before a disease had existed. Poisoned or not, there is no doubt that Burrus was ill of a natural disease. The disease must have had a chronic character, as is evident from the fact that the emperor had time enough to pay a visit to Burrus and to send him a medicament. No mention is made of a fever and so an acute affection of the throat can be excluded. Fever (calor praeter naturam) was very well known to the physicians of that epoch and could not escape their attention. The disease must have been accompanied by fetor from the mouth. This was the reason Burrus had to turn his face away from the emperor, much beloved on every other occasion. And the divine emperor was not offended by this gesture of his subject. He understood the conduct of the sick man. The few words pronounced by the sick indicate that he suffered pains in speaking, and so he was excused by the emperor for the lack of due reverence. These facts, together with the advanced age of the sick man, and with the description of his fatal illness, can hardly be interpreted in any other way than that Burrus succumbed to a laryngeal carcinoma. The assumption of poisoning is quite superfluous.

Many centuries later the first case of laryngeal carcinoma was found and described by Joseph Lieutaud (1703-1780), who reports in his "Historia anatomico-medica" on the autopsy of a man 50 years of age who during his life had lost his voice and had difficulties in swallowing. At the autopsy he had "many tumors in the pharynx and in the larynx showing the aspect of cancer."[7]

But the diagnosis was not easy. Gaspar Laurent Bayle (1774-1816), in his famous "Traité des maladies cancéreuses," which after his death was published by his nephew, A. L. J. Bayle, in Paris in the years 1833 and 1839, expressed the opinion that a laryngeal carcinoma can be detected only at autopsy and cannot be

4. G. Suetonius Tranquillus: Nero Claudius Caesar, Chap. 35.

5. Weigall, A. E. P. B.: Nero, the Singing Emperor of Rome, London, Putnam Butterworth, 1930.

6. P. Cornelius Tacitus: Annales XIV, 51.

7. Lieutaud, J.: Historia anatomico-medica, Paris Vincent, 1767.

diagnosed during life.[8] Thuillier gave a description of diagnostic procedures then in use: palpation of the larynx through the mouth and from the outside, intended to detect crepitation.[9]

The diagnosis of a laryngeal carcinoma in the living patient was not possible with certainty until the discovery of the English physician B. G. Babington, who presented to the Hunterian Society in London (March 1829 and described on page 555 of the *Medical Gazette* of London in the same year, under the name "Glotiskop") the first laryngeal mirror.[10] But it was not until the year 1854 that the laryngoscope was introduced into general use by the Spanish singer Manuel Garcia, an exceptional man who reached the age of 100 years.

Within a few years of the introduction of the laryngoscope, Baker[11] reported on five primary carcinomata of the larynx among 500 cancer patients—not only an unexpected finding but also a marvelous advance in diagnostic possibilities.

The possibility of making the diagnosis of a laryngeal carcinoma during life is an acquisition of recent times; no wonder the physicians of ancient Rome had not detected the tumor, thus starting the rumors of poisoning.

Gunduliceva 19.

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8. Bayle, G.-L.: Traité des maladies cancéreuses: Ouvrage posthume, precédé du portrait de l'auteur et d'une notice historique sur sa vie et ses ouvrages; Revu, augmenté et publié par son neveu, A.-L.-J. Bayle, Paris, Laurent, 1833-1839, Vol. 2, Bk. 4, Obs. 82.

9. Tuilier, J. B.: Essai sur l'angine laryngée œdémateuse, Paris, 1815.

10. Babington, B. G.: Glotiskop, Med. Gaz. p. 555 (March) 1829.

11. Baker, W. M.: Contribution to the Statistics of Cancer, Med.-Chir. Tr. **45**:389-406, 1862.

THIOPENTAL WITH SUCCINYLCHOLINE AND TOPICALLY APPLIED TETRACAINE

A More Ideal Anesthetic for Peroral Endoscopy

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GENERAL anesthesia for peroral endoscopy has always presented many problems. The use of inhalants, ether and/or cyclopropane, is time-consuming and fraught with the potential danger of explosion, due to a spark from the endoscope light, and of anoxic episodes. Furthermore, the maintenance of the desired level of anesthesia in prolonged procedures is difficult or impossible.[1]

The use of intravenous anesthetics eliminates the danger of explosion and facilitates the maintenance of the desired level of anesthesia, but it introduces new problems, i. e., prolonged postoperative depression and the serious danger of laryngospasm.[2] The latter has been obviated to a certain extent by preceding the general anesthesia with topical anesthesia and utilizing a curariform drug in combination with the intravenous barbiturate.[3] However, the addition of curare adds to the postoperative depression, thus necessitating exceedingly watchful postanesthesia nursing care, and increases the risk of postendoscopic complications.

The ideal anesthetic for peroral endoscopic procedures would be one which would embrace the following properties: Narcosis and good muscular relaxation could be attained for a short controlled period; it would allow both rapid induction

Succinylcholine chloride (anectine®) was provided by Burroughs Wellcome & Company, Inc., Tuckahoe 7, N. Y.

Dr. Herrmann E. Bozer, Chief of the Otolaryngological Service of the Buffalo General Hospital, provided cases for this study.

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1. Whalen, E. J.: Anesthesia in Peroral Endoscopy, Ann. Otol. Rhin. & Laryng. **53**:469-479 (Sept.) 1944. Moorhead, R. L.: Symposium on Peroral Endoscopy: Anesthesia, Laryngoscope **49**:1162-1164 (Dec.) 1939. Cullen, S. C., and Trapasso, A. J.: Use of Curare to Facilitate Endoscopy, Arch. Otolaryng. **38**:347-349 (Oct.) 1943.

2. Pembleton, W. E., and Vinson, P. P.: Anesthesia in Peroral Endoscopy, Arch. Otolaryng. **50**:561-563 (Nov.) 1949. Cooper, M. P.: Use of Pentothal Sodium Intravenously for Anesthesia in Laryngoscopy, Bronchoscopy and Esophagoscopy, Anesth. & Analg. **18**:181-185 (July-Aug.) 1939.

3. Silverberg, J. S., and Ansbro, F. P.: An Evaluation of the Use of Curare in Endoscopy, New York J. Med. **44**:2468-2471 (Nov.) 1944. Sircar, B. N.: Curare-Pentothal Anesthesia in Endotracheal Intubation and Endoscopy, J. Internat. Coll. Surgeons **17**:685-687 (May) 1952.

and rapid emergence, with a rapid return of pharyngeal and cough reflexes. There would be minimal respiratory and circulatory depression.

Since the intravenous route of general anesthesia preceded by topical anesthesia seems to offer many desirable features, in these respects the use of an especially quick-acting curariform drug in combination with the barbiturate would appear to offer a definite advantage.

Consequently, succinylcholine chloride was used in combination with thiopental sodium anesthesia in order to approach more nearly the ideal general anesthesia technique for peroral endoscopy.

PROCEDURE

All patients to undergo peroral endoscopy (laryngoscopy, bronchoscopy, or esophagoscopy) were given morphine and scopolamine or atropine in the ratio of 25:1 one hour preoperatively. Each patient was thoroughly anesthetized topically with 2% tetracaine (pontocaine®) hydrochloride 10 to 15 minutes prior to endoscopy. This was begun with three gargles of 2 cc. each at two-minute intervals, followed by swabbing the piriform sinuses with cotton soaked with 2% tetracaine and then by spraying 2 cc. of 0.5% tetracaine into the glottis through a blunt, curved cannula placed in the oropharynx.

The patient lay on the table with the shoulders just above the break, so that the endoscopist's assistant could more effectively control the position of the head. An intravenous infusion of appropriate fluid was started with a slow rate of flow. Preliminary pulse rate, respiration rate, and blood pressure determinations were recorded. The endoscopist checked his instruments for adequate illumination and the suction apparatus for proper functioning before the induction of anesthesia. The patient was then given 200 to 300 mg. of thiopental sodium, followed in 60 seconds with 20 to 40 mg. of succinylcholine chloride. The endoscopist was instructed to proceed after 30 seconds. Nasopharyngeal administration of oxygen was started immediately after giving of thiopental, with a flow of 8 liters per minute. Increments of thiopental and succinylcholine chloride were added as deemed necessary to control movements by the patient and to provide adequate relaxation of the jaw and the pharyngeal and glottic musculature. Unless the patient demonstrated an extremely light plane of anesthesia, as evidenced by grimacing and/or movements of the extremities, additional thiopental was not used, so that the patient would awaken as soon as possible after completion of the procedure. If necessary, thiopental sodium was added in 50 mg. increments. When more relaxation was required, succinylcholine chloride was added in 10 mg. increments. Vital signs and color were closely followed and were recorded at the completion of the procedure. Suction and oxygen were utilized as indicated. An anesthesia machine was available at all times during the procedure, so that resuscitation could be rapidly and efficiently performed if apnea occurred.

RESULTS

Fifty consecutive endoscopic procedures were performed with the aforementioned technique. These were distributed according to procedure as follows: laryngoscopy, with or without biopsy, 19; bronchoscopy, 8; esophagoscopy, 16, and combined, 7. The age distribution of the patients in this series ranged from 20 to 75 years.

Dosage of thiopental sodium and succinylcholine chloride was in accordance with the data given in the charts. It is apparent that a large number of the procedures were performed without additional drugs, other than the initial 300 mg. of thiopental sodium and 30 mg. of succinylcholine chloride. Topical anesthesia, in the opinion of the anesthesiologist, proved unsatisfactory in two cases in this series.

Usually patients reacted to painful stimuli within two minutes and to spoken commands within four to five minutes following completion of the examination or procedure.

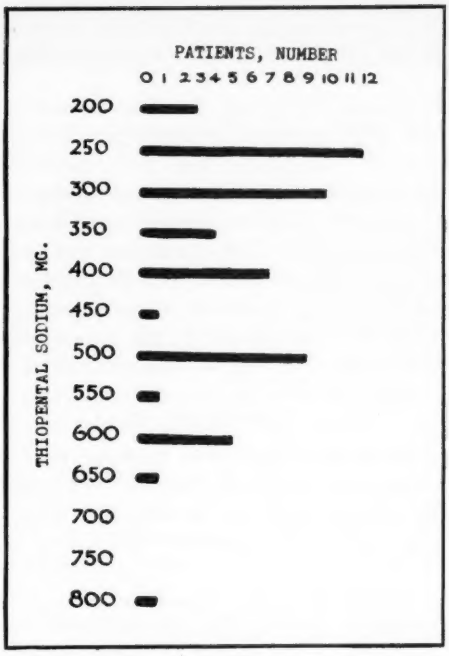

Chart 2.—Succinylcholine chloride dosage.

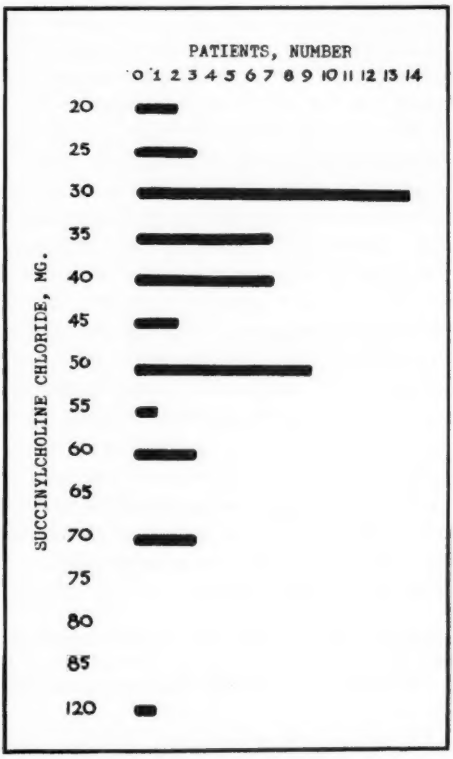

Chart 1.—Thiopental sodium dosage.

No adverse reactions have been noted. In a few instances, a slight rise in blood pressure resulted from slight straining by the patient or beginning hypercapnia (hypercarbia).

Within five minutes after completion of the procedure, most patients were awake and showed no signs of respiratory or circulatory depression.

Apnea necessitating interruption of the procedure and institution of resuscitation with bag and mask occurred in three cases. Respirations returned promptly, within three to five minutes. No instances of prolonged apnea, as reported by Harper [4] and by Love,[5] were noted. However, the dose of succinylcholine chloride exceeded 70 mg. in only one case, in which 120 mg. was given.

COMMENT

While one can attain excellent degrees of muscular relaxation, without apnea in most cases, with deep ether anesthesia, the induction period is markedly prolonged, even when the technique proceeds smoothly. A 20-to-30-minute induction time seems incongruous indeed for a procedure which may last only two to five minutes.[1]

In many instances, the patient to be examined by endoscopy has already demonstrated respiratory difficulties, and the induction period with ether, with the additional secretions it produces, is endangered by coughing and obstruction, which might lead to recurrent anoxic episodes. For these reasons, many anesthesiologists have abandoned ether as the anesthetic of first choice in endoscopy.

Cyclopropane has been avoided by many anesthesiologists for endoscopy because of its explosiveness and the rapid emergence with this agent. While adequate relaxation may be attained with a rapid and usually smooth induction, the equally rapid emergence does not allow adequate time for endoscopic examination. Concern has been expressed over the sensitization of vagovagal and sympathetic reflexes occurring during endoscopy with the production of marked arrhythmias and cardiac arrest. Thiopental alone does not provide adequate relaxation of the jaw or abduction of the vocal cords without the use of bludgeoning doses. The circulatory depression as well as the respiratory depression occurring with large doses of thiopental are poorly tolerated by many patients subjected to endoscopic procedures.[2]

Thiopental-curare anesthetic combinations carry the same objection for endoscopic examination. However, smaller doses of thiopental can be used here, and adequate relaxation is obtained.[3] Marked respiratory depression with concomitant anoxia is frequently seen. While the induction period is short, postoperative depression persists for a considerable period after the short endoscopic procedure is completed. Pharyngeal reactions are dulled or absent, and these patients cannot handle blood or secretions in an already compromised airway. Analeptics have been resorted to in an effort to shorten the period of postoperative depression, and, while these may temporarily arouse the patient, secondary depression after the patient has returned to his bed may be of serious extent.

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4. Harper, J. K.: Prolonged Respiratory Paralysis after Succinylcholine, Correspondence, Brit. M. J. **1**:866 (July) 1952.

5. Love, S. H. S.: Prolonged Apnœa Following Scoline, Correspondence, Anæsthesia **7**:113-114 (April) 1952.

The contraindications for the use of thiopental *per se* pertain to the poor-risk patient who requires endoscopy; however, the use of the topical technique alone usually proves quite satisfactory in these patients.

Succinylcholine chloride seems to aid in approaching the ideal endoscopic anesthetic by providing relaxation for short intervals of time.

Succinylcholine chloride, or diacetylcholine, is chemically succinic acid bis (beta-dimethylaminoethyl) ester dimethylchloride. It causes muscular paralysis by producing a blockade of nervous transmission at the myoneural junction. Repeated injections produce reproducible and predictable muscular paralysis. Neither tachypylaxis nor significant cumulative effects have been seen. Its effect is not changed by previous doses of decamethonium bromide or tubocurarine chloride.[6]

No significant changes in blood pressure or histamine-like depressor action have been noted following its use. Succinylcholine chloride is hydrolyzed by cholinesterases, and this accounts for its extremely short duration of action. Liver or kidney pathology does not preclude its use. Rapid injections of succinylcholine chloride may produce muscular fasciculations due to the initial stimulating action of the drug. Such fasciculations have been noted at the start in some of the cases in this series.

The typical course of an endoscopic procedure utilizing succinylcholine chloride was seen in the following case.

A 54-year-old husky, short-necked white man, with a history of chronic cough and hoarseness for a four-month period, was brought to surgery for laryngoscopy and biopsy.

He had been given morphine sulfate, 0.010 gm., and scopolamine hydrobromide, 0.0004 gm., one hour before. He was locally anesthetized with 2% tetracaine hydrochloride gargle and swabbing and 0.5% spray. After a 15-minute period he was positioned on the operating room table. The laryngoscope lights were checked, an infusion was started, suction was connected. The surgeon and nurse were ready to proceed immediately. An oxygen catheter was placed in the nasopharynx with 8 liters of oxygen flowing per minute. Thiopental sodium, 250 mg., was given slowly, followed by 30 mg. of succinylcholine chloride in a separate syringe.

The blood pressure and pulse are checked prior to and during the procedure. A finger is kept on the radial pulse throughout the procedure. Apnea and jaw relaxation are generally present 10 to 15 seconds following succinylcholine chloride injection, and the endoscopist is then asked to proceed. Apnea, if present, usually lasts 45 to 60 seconds. The procedure may take approximately three to five minutes. Increments of 50 mg. of thiopental sodium or 5 mg. of succinylcholine chloride are given as needed. The patient usually reacts quickly, within three to five minutes after the procedure is completed, and if less than 500 mg. of thiopental sodium is given, he will be moving around, coughing, and even reacting to questions. He is well able to handle any blood or secretions in his pharynx.

With a skilled endoscopist the entire procedure from induction to recovery has never been longer than 7 to 10 minutes. Adequate relaxation and narcosis have been provided for the optional time necessary to accomplish the procedure.

It is apparent to us that the distinctive advantage of succinylcholine chloride over the other curariform drugs is its short duration of action. The short period of

6. Castillo, J. C., and de Beer, E. J.: The Neuromuscular Blocking Action of Succinylcholine, J. Pharmacol. & Exper. Therap. **99**:458-464 (Aug.) 1950. de Beer, E. J., and others: Synthetic Drugs Influencing Neuromuscular Activity, Ann. New York Acad. Sc. **54**:362-372 (Oct.) 1951.

apnea, when it has occurred, has not proved troublesome, resuscitation with bag and mask being instituted promptly.

The rapid return of consciousness and cough reflex allows the patient to handle blood and secretions more adequately. This is particularly important when bronchial and laryngeal biopsy specimens need to be taken under general anesthesia. The rapid return of full and vigorous respiration obviates the dangers of prolonged intercostal paralysis.

We have been able to perform most of these procedures with minimal doses of thiopental and succinylcholine chloride, rapid endoscopy making this possible. Even in those cases in which the procedure was prolonged to between 20 and 30 minutes, the doses of succinylcholine chloride did not reach high enough levels to produce cumulative effects and prolonged apnea. The value of a short-acting curariform drug in prolonged procedures lies in the possibility of using repeated doses without great concern over cumulative effects and postanesthetic depression.

Part of the ability to utilize minimal doses of thiopental and succinylcholine chloride lies in the adequate topical anesthetization of the pharynx and larynx, so that the patient does not react to endoscopy in light anesthesia. During the period that succinylcholine chloride was used for endoscopic procedures it was noted that the drug was equally useful in short procedures of other types.

Orthopedic manipulations and closed reductions of fractures and dislocations present a similar set of requirements for the exacting anesthesiologist, namely, moderately deep narcosis and good relaxation for a short time. The combination of small doses of thiopental and succinylcholine chloride seems to us to approach these requirements most closely. It has also been useful in short operative procedures, particularly about the head and neck, in which endotracheal intubation has been deemed advisable.

SUMMARY

Fifty endoscopic procedures were performed with the anesthetic combination of thiopental, succinylcholine chloride, and topically applied tetracaine.

Most of these procedures were performed with minimal doses of thiopental sodium ranging from 250 mg. to 400 mg., and of succinylcholine chloride, ranging from 25 to 40 mg.

It is our opinion that this combination of drugs most nearly fulfills the requirements for an ideal anesthetic for peroral endoscopy. Its chief advantages lie in the rapid return to consciousness of the anesthetized patient and the return of adequate activity of the respiratory musculature with a concomitant rapid return in ability to handle the secretions and blood incidental to endoscopic procedures.

PREANESTHETIC SEDATION OF CHILDREN

Analysis of the Effects for Tonsillectomy and Adenoidectomy

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THE NECESSITY for preanesthetic sedation before tonsillectomy and adenoidectomy in children is a much debated question. Many otolaryngologists avoid giving sedation because of the danger of depression of the cough reflex and the attendant hazard of aspiration of blood and debris into the lungs. On the other hand, pediatricians and psychiatrists believe that undesirable personality changes may be related to inadequate preoperative preparation and sedation. The anesthesiologist finds himself in a dilemma; he does not wish to add to the risks of operation and anesthesia, yet wants to avoid the unpleasant task of anesthetizing apprehensive, struggling, or crying children. The investigation reported herein was begun in order to obtain answers to such problems. Is the incidence of crying (and presumably the danger of psychic trauma) higher in children given only a belladonna drug before anesthesia? Does the employment of sedatives increase the hazards of anesthesia and operation?

METHOD OF STUDY

Questionnaires attached to the anesthetic records were filled out by the anesthetist during the anesthesia. The data from 1,500 children operated upon for tonsillectomy and adenoidectomy were collected from the anesthesia services of three hospitals.[1] Anesthesia was administered in most instances by physician anesthetists and in the remainder by medical students or nurse technicians supervised by staff anesthesiologists.

Several combinations of drugs were used for preanesthetic medication, as indicated in Tables 1 to 4. Pentobarbital in a sweet syrup was given by mouth. The dosage scale recommended by Leigh and Belton[2] was used. Patients given rectal basal narcosis have not been included in this series.

Anesthetic agents employed for induction of anesthesia included vinyl ether (vinethene®), ethyl chloride, nitrous oxide-cyclopropane, and in case of several older children thiopental by intravenous injection. The open method of inducing anesthesia was used in approximately 75% of the patients, and a to-and-fro semiclosed system with nitrous oxide-cyclopropane in the remainder. Anesthesia was maintained with ether-oxygen by oropharyngeal insufflation in 50% of the patients and by the endotracheal route in the remainder.

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1. Hospital of the University of Pennsylvania, Children's Hospital of Philadelphia, and Graduate Hospital of the University of Pennsylvania.

2. Leigh, M.D., and Belton, M. K.: Premedication in Infants and Children, Anesthesiology **7**:611-615, 1946.

RESULTS

Time of Preanesthetic Medication.—Despite the recommendation that preanesthetic medication be given one hour prior to the induction of anesthesia, a minority of otolaryngologists persisted in ordering such drugs "on call." This usually meant that the child received his injection a few minutes before induction of anesthesia. For this reason and because of delays in starting operations, in one child of four either the maximal benefit from the medication had not yet been achieved or the effect of the drugs was already wearing off before operation.

Incidence of Crying.—As might have been expected, crying was commonest in children 5 years of age or younger (Tables 1, 2, and 3). The incidence was markedly influenced by preanesthetic medication. It was highest when a belladonna drug was used alone and least when a combination of pentobarbital, morphine, and belladonna drug was administered. Scopolamine was found to have a better sedative effect than atropine. It was apparent that more careful attention to the time of administration of sedatives would have reduced the incidence of crying. This is exemplified by the fact that 40% of crying children under the age of 5 years had not been given medication on time, whereas only 10% of the noncrying children had failed to receive medication within the specified interval.

It was of considerable interest that the incidence of anesthetic complications was higher in the crying children than in the noncrying ones (Table 4).

Side-Effects of Preanesthetic Medication.—Several children were considered to be more depressed than desirable with the combination of belladonna, opiate, and barbiturate drugs. However, in no instance was it necessary to postpone operation because of depression, and none of these children gave cause for alarm in the postoperative period.

It was noted that after the injection of scopolamine, the skin of the child frequently felt hot; hence, the rectal temperatures of 102 children who had received scopolamine alone or in combination were measured. In seven children there was an elevation of body temperature above that recorded before the scopolamine was given. In each of these seven, the rise was less than 1 degree Fahrenheit.

Evidence of mild agitation was noted in 24 children, all of whom had had scopolamine. Twenty of these were crying children in whom muscular activity seemed unusually prominent. In the other four, muscular activity without crying seemed excessive. None of the patients was sufficiently affected to cause concern.

Circumoral pallor was noted in 23% of the children given scopolamine and in 10% of those given atropine. No particular significance was attached to this.

Induction of Anesthesia.—Vinyl ether was used most frequently for induction (596 patients), with a change to ethyl ether when the first plane of anesthesia was reached. Ethyl chloride was employed for induction in similar fashion in 478 patients. To 78 children, ethyl chloride was given until consciousness was lost, followed by vinyl ether, which, in turn, was succeeded by ethyl ether. The remainder of the patients received nitrous oxide and/or cyclopropane until the first plane of anesthesia had been reached, after which ethyl ether was added to the mixture in the anesthetic bag or administered by an open technique.

The incidence of respiratory depression during induction of anesthesia was greater after the use of scopolamine and barbiturates and/or opiates for preoperative medication. Severe depression was not encountered, and moderate respiratory

TABLE 1.—*Effect of Preanesthetic Medication on Incidence of Crying in Children of Less Than Five Years*

Agent	Patients	
	No.	% Crying
Atropine	77	39
Scopolamine	33	36
Pentobarbital and atropine	34	18
Pentobarbital and scopolamine	289	17
Pentobarbital, morphine, and atropine	9	11
Pentobarbital, morphine, and scopolamine	163	15

TABLE 2.—*Effect of Preanesthetic Medication on Incidence of Crying in Children of Six to Ten Years*

Agent	Patients	
	No.	% Crying
Atropine	49	16
Scopolamine	25	8
Pentobarbital and atropine	14	9
Pentobarbital and scopolamine	233	6
Pentobarbital, morphine, and atropine	14	36
Pentobarbital, morphine, and scopolamine	225	5

TABLE 3.—*Effect of Preanesthetic Medication on Incidence of Crying in Children of Eleven Years or Older*

Agent	Patients	
	No.	% Asleep *
Atropine	8	0
Scopolamine	2	0
Pentobarbital and atropine	6	0
Pentobarbital and scopolamine	27	11
Morphine and atropine	10	0
Morphine and scopolamine	25	8
Pentobarbital, morphine, and atropine	7	0
Pentobarbital, morphine, and scopolamine	126	16

* Crying not used here, because only one child in the entire group cried. "Asleep" means sleeping in bed or on the litter while awaiting operation.

TABLE 4.—*The Crying Child and Anesthetic Complications*

	Asleep or Not Crying, %	Crying, %
Respiratory depression, induction	5.0	6.0
Respiratory depression, maintenance	4.0	6.0
Cyanosis, induction	8.0	14.3
Cyanosis, maintenance	11.0	21.0
Vomiting, induction	2.0	7.0
Vomiting, postoperative	30.0	38.0

depression was noted only in the groups given scopolamine with pentobarbital and/or morphine. As might be anticipated from this, the incidence of cyanosis during induction of anesthesia was greatest when scopolamine combinations had been administered. Only one child given atropine alone became cyanotic during induction of anesthesia, whereas 14% given scopolamine alone became cyanotic. The only instances of severe cyanosis were observed in patients given scopolamine. It should be noted that there were five times as many patients in the scopolamine groups.

Vomiting during induction of anesthesia was commonest when atropine or scopolamine was given as the sole preanesthetic drug (5 and 6%, respectively). With combinations of medication, approximately 2% of the children vomited.

In nine children convulsive movements developed during induction of anesthesia. The convulsions were mild and terminated promptly, and all were associated with vinyl ether anesthetic inductions. Vinyl ether was administered to 674 patients (vinyl ether-ether plus ethyl chloride-vinyl ether groups) and was not given to 809 children. In one patient, twitching movements were confined to an arm and hand; in another, to a shoulder; in the remainder, convulsions were generalized. All occurred after vinyl ether had been given for several minutes or just after the change to ethyl ether had been made. A mild degree of cyanosis often preceded the convulsion but was not noted in all patients. Treatment consisted in discontinuing anesthesia and administering oxygen. Subsequently, the operation was done, and none of the patients showed evidence of untoward postoperative complications.

Postoperative Course.—All patients were observed in the operating room or recovery room until pharyngeal and laryngeal reflexes returned and there was response to name or stimuli. Preanesthetic medication other than atropine or scopolamine delayed full emergence from anesthesia. No postoperative or postanesthetic complications were noted in these patients. It may be of significance, however, that the endotracheal route of maintaining anesthesia was employed in most children given scopolamine, morphine, and pentobarbital. Children given atropine or scopolamine alone seemed to awake earlier, moved in their beds, and cried, whereas the children given the combinations of drugs tended to lie quietly in bed for two to four hours, occasionally coughed and moved, but did not cry often.

COMMENT

Too often, tonsillectomy and adenoidectomy have been considered a necessary evil of growing up. The child's contact with hospitalization and operation has been looked upon in terms of adult experience. The average child knows little of what to expect unless his parents have pictured what may happen to him. He may be greeted in the hospital by people who stick him with needles, wear masks, and cause him to breathe disagreeable odors that result in loss of consciousness. He frequently does not understand why. Psychiatrists have demonstrated that undesirable personality changes sometimes occur subsequent to hospitalization and operation. This occurrence is especially evident in small children, its incidence tapering off with advancing age and/or improved understanding of the situation.[3] In spite of this,

3. Levy, D. M.: Psychic Trauma of Operations in Children and Note on Combat Neurosis, Am. J. Dis. Child. **69**:7, 1945. Coleman, L. L.: The Psychological Implications of Tonsillectomy, New York J. Med. **50**:1225, 1950.

preoperative sedation is often denied the child. Adults, better able to withstand insults to the psyche, are routinely given opiates, belladonna drugs, and often barbiturates.

The data presented emphasize the fact that the small child is the one most disturbed by operation and anesthesia. The figures further show what can be accomplished by the addition of pentobarbital with or without morphine. More attention to the time of administration of medication could further reduce the incidence of crying. Rectal basal narcosis may abolish crying, but this has not been employed routinely because the increased time and personnel required have not been available.

Considered alone, the facts presented argue that sedatives be given before children are anesthetized. If, however, a decrease in the incidence of psychic trauma is gained at the expense of an increase in postoperative morbidity, then the danger of psychic trauma should be minimized. The fear that preanesthetic sedation may increase the risk of respiratory depression during the period of operation is supported by these data. It should be recalled, however, that in this series anesthesia was administered by physicians and nurses of varying experience in administering anesthetics. When experienced physicians administer the anesthetics, respiratory depression might not be encountered. It is probable that with inadequately trained anesthetic personnel, psychic trauma is of secondary importance and use of sedatives should be avoided.

Evidence of undesirable depression in the postoperative phase could not be elicited when sedation had been employed before anesthesia. The fact that children who had been given combinations of barbiturates, morphine, and belladonna drugs tended to sleep quietly postoperatively was not thought to be undesirable, provided active pharyngeal and laryngeal reflexes were present. The fact that the latter reflexes appeared to return as promptly with sedation as without sedation may be accounted for by the fact that less anesthetic agent may have been needed when sedation had been employed. It bears reiteration that these children were carefully watched in the immediate postoperative period, either in the recovery or in the operating room. Without careful observation, preanesthetic sedation should probably be curtailed.

The data reported suggest that there is little to be gained in the use of morphine as an addition to preanesthetic medication. The sedative effect when added to that of a barbiturate appeared to be negligible. In addition, it seemed that when opiates had been given and oropharyngeal insufflation anesthesia used respiratory drive was diminished whenever partial respiratory obstruction occurred. This factor was relatively unimportant when the anesthetic was administered by the endotracheal route. The conclusion appears inescapable that when oropharyngeal insufflation anesthesia is used, morphine is contraindicated and that its use otherwise in otolaryngological operations is dependent upon the skill of the anesthesiologist.

The response to injection of scopolamine has been revealing. The data presented contain ample evidence of the sedative effects of scopolamine. They also indicate a surprising degree of respiratory depression. There are data [4] to indicate that scopolamine can depress respiratory activity in some people, stimulate it in others,

4. Loeschcke, H. H., and Wendel, H.: Die Wirkung von Morphin, von Scopolamin und ihrer Kombination auf die Lungenbelüftung beim Menschen, Arch. exper. Path. u. Pharmakol. **215**:241, 1952.

and be without effect in a third group. The respiratory-depressant effects of scopolamine may be additive to those of morphine.[4] The action of scopolamine in raising body temperature in a small percentage of patients suggests the advisability of decreasing the dosage in hot and humid weather.

The method chosen to induce anesthesia appeared relatively unimportant. The anesthetist should be aware of the convulsant properties of vinyl ether and be prepared to deal with them. Anoxia should be avoided, although cyanosis was not always detected prior to convulsions. Ethyl chloride does not appear to be an agent that can be summarily dismissed as intimated by some.[5] Properly utilized, it may be equal to and possibly superior to vinyl ether for induction of anesthesia by the open technique.

Obviously, skill in administering anesthetics is of paramount importance. A physician versed in the art of pediatric anesthesia can anticipate the difficulties and complications presented in this report and prevent them or minimize them should they occur. On the other hand, the occasional pediatric anesthetist, unaware of the problems peculiar to children, may encounter troubles of even greater magnitude than discussed herein.

SUMMARY

The data obtained from a study of the effects of preanesthetic medication on 1,500 children before tonsillectomy and adenoidectomy have been presented. From these data it is apparent that (1) the incidence of crying prior to anesthesia can be sharply reduced by the addition of a barbiturate to the belladonna drug; (2) crying increases the hazard of anesthesia; (3) respiratory depression and cyanosis appear more frequently if morphine and pentobarbital are included in preanesthetic medication, but no evidence was obtained that reflexes are depressed unduly postoperatively; (4) scopolamine is a better sedative than atropine; and (5) morphine should not be employed in preparation for this operation, but a barbiturate can and should be administered.

Margery Van N. Deming, M.D.; H. I. Stone, M.D., and Angela Verratti, R.N., assisted in collecting the material from which this paper was written.

5. Stephen, C. R.: Operating Room Fatalities in Pediatric Surgery, Anesth. & Analg. **31**:36, 1952. Adriani, J., and Bush, O.: Hazards of Ethyl Chloride Anesthesia, New Orleans M. & S. J. **104**:427, 1952.

"I SCARCE WAS SURE I HEARD YOU"

Saga of the Work in Prevention, Conservation, and Rehabilitation by and for the Hard of Hearing

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"I scarce was sure I heard you."
—Edgar Allan Poe.

THIS LINE of Poe's was chosen for the title of my paper because it well serves to distinguish the state of the hard of hearing from that of the deaf. I am concerned here primarily with the hard of hearing; the deaf and schools for the deaf will be mentioned only incidentally.

It is an honor to be chosen by the Otosclerosis Study Group to tell the story of the birth and growth of the work of organizations for the hard of hearing. I trust that my treatment of the subject will convey more than a tale of struggle and mounting success, rather a story of valiant endeavor by the hard of hearing in behalf of the hard of hearing, of their contributions to otology by teaching, research, understanding, and, above all, constructive thinking and example.

For a better understanding of some of the problems involved, a few words should be said about the mysteries of the phylogenesis of the ear, hearing in general, and the philosophy of rehabilitation, or adjustment to deafness.

Hearing is in essence a sensation of shearing touch or pressure. No matter how lowly, all animals, and in a sense even some plants, possess an ability to hear if by hearing is implied an ability to react to vibrations in their immediate environment. However, only terrestrial fauna possess organs specifically developed to hear and differentiate air-borne sounds.

Countless eons ago our prehistoric ancestors, living in the sea and in the vaporous or steaming ooze of the tidal wash along its shores and indentations or in inland water pools large and small, slowly evolved organs of hearing from what at first was a general surface, or skin-touch, apparatus. This apparatus eventually became localized and transformed into a small organ on the surface of the body. This organ (this primitive ear) was sometimes on the head, sometimes on the back or tail, and sometimes, as in certain insects, on the feet or other parts of the body.

The structures forming the ear of man and the higher vertebrates have been traced phylogenetically, with large gaps, down to the primitive forms, as in the jellyfish. These structures are called "anlages." They were at first useful only for sensing underwater vibrations, and so our primordial progenitors, like the

Read before the combined meeting of the Committee for the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology and the Otosclerosis Study Group.

present-day fishes, lived in a relatively silent world, although they sensed pressure variations and certain species possessed some discrimination of vibration. Living under water they could not sense air-borne sounds. To quote again from Edgar Allen Poe, "The quiet was unbroken, and the silence gave no token"—no token of the presence of terrestrial friend, foe, or food. There was no sound if by sound we mean audible vibration, because air-borne sounds are reflected from the surface of water; they do not reach the depths. With the slow lapse of ages, as the seas receded and land masses and terrestrial animals appeared, more predatory foes with or without legs or wings were on the prowl and more and more it became necessary for survival and for the perpetuation of the species to hear air-borne sounds: to avoid danger, to sense the presence and localization of prey, and to hear the mating call of the opposite sex. (Some cynic might call the last courting rather than avoiding danger!)

Few individuals or species survived without possessing at first good sensation for water-borne and eventually good hearing for earth- and air-borne sounds. Good hearing was a large factor in the survival of the fittest, although the other senses played important roles. In many terrestrial animals, including mankind, although eyesight, touch, and smell complicate the picture, the love song above all still is heard throughout the land, mankind seemingly being the only animal to distort it deliberately into the dissonance of "bee-bop" or "bee-bo" or be-something else equally disagreeable to a sensitive ear.

It is usually said that in man there are five senses—sight, smell, taste, touch, and hearing—all of which may be useful in certain types of communication. To these I would add body coordination and orientation in space and movement. Two of the sense organs, those of equilibration and of hearing, are housed in the chambered capsule of the inner ear, and these two even in the higher forms are still basically similar in function. One senses motion (and absence of motion) of the head and body; the other senses motion (and absence of motion) in the environment, whether it is water, earth, or air. When motions in the environment cause a sensation of sound, they are called sound waves or vibrations.

All the senses with the exception of hearing and sight are concerned mostly with the immediate environment; in other words, more with near than with far factors. However, while the eyes are closed sight ceases, and when the ears are blocked sounds are less readily detected.

Hearing is a social function. It is a very important social function but it is not an absolute social necessity. It is not a guarantee of happiness; many persons flourish without it and many wither who possess it. Of course absence of both speech and hearing is much more distressing than a loss of either alone.

There are signs, that in the not-so-distant future, because of lessening importance for survival, both speech and hearing in man may gradually deteriorate. Will this be good or bad? Who knows?

More than the ear is necessary for good hearing because for all sense organs there is a central (or psychic) apparatus without which the sense in question produces only an automatic, reflex, low-level, or unconscious response valuable for alarm reactions directly to the muscles of the body but useless for fine discriminatory functioning. When you jump from a loud noise even before you are aware you have heard it, that is an example of an alarm reaction.

To understand speech or other sounds fully we must employ more than our outer, middle, and inner ears; we must possess and use both central and peripheral analysis; we must use our brains as well as our ears. This fact is one reason why so-called exact, or observational, research is unable to give us all the answers. An absolute value cannot be assigned to any biologic measurement. There is no standard pattern of response; there is only a range of variables. Psychic response is often unpredictable and unstable. The old saying "Grandma can hear when she wants to hear" still often holds true. Remember that one may consciously and deliberately ignore a stimulus. I once knew an old rascal who would not admit he heard a sound unless a pretty girl put her lips close against his ear. He got a great kick out of this. You see, there are compensations even for severe deafness if you have a little imagination and use it.

The hard of hearing have always been aware of the varying and unpredictable responses to sound stimuli in deafened persons. They have demonstrated throughout the years that in many instances mere amplification of sound was not enough to enable them to hear. They have in their organizations provided the people, the living laboratories, in which research has resulted in a better understanding of the problems involved. They have been living examples of the effect upon the hearing of the many disorders which affect the mechanical and neural mechanisms of the ear.

The hard of hearing themselves have been the pioneers in what is now called "audiology." From the first they have spread the gospel of early detection of deafness in the adult and, especially, in the child; they have stressed the importance of frank acknowledgment and early diagnosis. Without diagnosis there can be no prognosis, and without prognosis there can be no satisfactory plan for management and treatment.

Incidentally, some people forget that by diagnosis the otologist means determining which of several diseases or disorders is causing the symptoms and the pathology, not simply the amount and pattern of the hearing loss. It is stressed that even early detection, diagnosis, medical and surgical treatment, and follow-up are not enough. Continuing research is imperative.

Before the 20th century there were probably as many deafened people in the world in proportion to the population as today, but only the very deaf, or the "stone deaf," were really cared for. Institutions in those days taught, and some even today teach, only the sign language and not phonetic speech and not what is today usually called "lip reading." This practice tended to limit the social world of the severely deafened to their schoolmates and those few other persons who knew the sign language. The only efficient means of aiding the hearing was some form of speaking tube or trumpet which, although it did not amplify the voice, did channel the voice of the speaker directly into the ear of the hearer. It had other valuable assets: It excluded unwanted and confusing sounds; it was rugged and it had no movable or easily breakable parts; it was usable in all forms of deafness unless there was a total inability to hear speech. The speaking trumpet is still a useful instrument in very severe deafness. It is inadequate for distant speech. It is unsightly and a nuisance. Usually Grandma could hear without it if she really tried, and so she used her cupped hand and put off using the trumpet or any other hearing aid

until her family and friends almost lost their voices trying to make her hear them. For years Grandma could be a trial to her friends and to herself.

But grandmas, and younger people too, need not and many did not permit deafness to upset their lives. In the organizations for the hard of hearing we proved that many people with severe deafness could become adjusted to their deafness and even better adjusted to life than millions who possessed perfect hearing. Every man must learn to live with his limitations.

It may be said that "He has not lived who has not suffered pain, sorrow, and frustration" and learned how to live with himself. Otherwise he knows not the destiny of man. He is not mature. He is not grown up. The hard of hearing have suffered, they have lived, and many have grown up. Those who have not may at any time do so by facing facts and availing themselves of the aids now available.

Even before the turn of the century a few brave and venturesome souls began to gather in little groups here and there in the larger cities, some purely for human contacts, some to exchange opinions concerning their deafness and head noises, and some to have what they thought would be a receptive audience for their emotional outbursts. In such groups there were a few, a very few, mature and earnest men and women who wanted to help the hard of hearing to help themselves. Otherwise there was little interest in deafness in the first decade of the 20th century, although other handicaps received much attention, particularly blindness, paralysis and physical deformities.

The deafened, whether speechless or not, were listed under the category "deaf and dumb," "dumb" connoting not only inability to hear and speak but stupidity. In 1938, under the urging of the American Hearing Society, the United States Civil Service was persuaded to change its designation from "deaf mutes" to "the hard of hearing." It is clear who had been the "deafer and the dumber."

The deaf were often the butt of crude and cruel humor off and on the stage, and this fact was resented by some deafened people, their resentment perhaps starting the trend which now would taboo any humorous references to what are sometimes thought to be amusing characteristics of the Irishman, the Negro, the minister or priest, the Quaker, the Shaker, the Jew, or the Christian. Protests were made even in the silent-film days in regard to certain jokes about deafness. What a mistake this was! The deafened themselves found out that the only person who approaches emotional stability is he who can laugh at himself, and today organizations for the hard of hearing put on skits illustrating the very funny things that happen because of deafness, and the people who enjoy these the most are the hard of hearing themselves. This fact is one of the things I tell my patients.

One of the greatest deterrents to the formation of organizations for the hard of hearing has been a dislike, aversion, timidity, or cowardliness on the part of some hard-of-hearing persons, their families, and some of their friends most able to help financially to be publicly associated with the hard of hearing. They wanted to hide their deafness and their relatives' deafness, and they imagined they could do so because they wishfully tried to think that deafness is invisible. As a matter of fact, deafness is invisible only so long as nobody speaks, expects an answer, receives none, or receives one based upon faulty hearing. Deafness is frequently even more apparent than blindness. The minute you speak to me and it is evident

that "I scarce was sure I heard you" my deafness is no longer invisible. Why attempt the impossible? This philosophy is taught best by those who are hard of hearing; they are living examples of its efficacy. Some hearing-aid manufacturers and salesmen have been guilty of perpetuating the understandable desire of the timid and ill adjusted to hide deafness by overemphasizing in their advertising what they call "invisible hearing aids." This type of advertising should be discontinued.

Many deafened people feel that their associates are irritated by their deafness, but friends and relatives are irritated by some hard of hearing persons not because they do not hear well but because their reactions and behavior are often strange, overemotional, and even hysterical. The hard-of-hearing teachers in the chapters of the American Hearing Society were the first to understand and correct such reactions and to inculcate a proper philosophy in their pupils. One reason why the most successful teachers of the hard of hearing are usually hard of hearing themselves is that they are a living unanswerable argument to those who say that all the emotional and other troubles of the hard of hearing are caused by their deafness. These teachers are far better adjusted to life and its inevitable emotional episodes than many of the teachers with perfect hearing holding forth in our schools. They have profited by a proper understanding of their possibilities and limitations. It is an interesting fact that in some people deafness serves more as a stabilizing than as an upsetting factor. The motto of such people is "Adapt to deafness, do not fight it." Do not fight your hearing aid; learn how to use it.

A proper philosophy is important not only for a satisfactory life because it lessens excessive emotional reactions and protects the ear from neurovascular anoxias. It is therefore important as a prophylactic measure in several forms of deafness.

The pioneer in organizing the hard of hearing in this hemisphere was Edward Nitchie, who, beginning in 1903, gradually built up a group which in 1911 was called "The Service League" and in 1912 was incorporated. It had one paid social worker, Miss Anetta W. Peck. It was wholly supported by volunteer contributions and membership dues. This organization started the ball rolling and attracted the cooperation of a few otologists. (There were a few otologists, even in those days.)

Within two years the Nitchie group expanded, was renamed, and was chartered as the New York League for the Hard of Hearing. At first it had only one paid worker, 20 volunteer workers, a capital of $400, a blackboard, some hand-me-down furniture, and worn packs of cards. From the first the plan was not only to provide meeting places for the hard of hearing but to improve their social happiness and economic status and their opportunities for self-advancement and employment, to investigate and advise in regard to the proper choice and use of hearing aids (1922), to protect the hard of hearing from being fooled by misleading advertising, and to steer them away from quackery of all kinds. Surveys were instituted [1] for the purpose of detecting and diagnosing hearing losses in children, by medical follow-ups and teaching to help hard of hearing children to continue in regular schools instead of being segregated in schools for the deaf, and to educate not only the children but

1. The first audiometric and medical survey of the hearing of school children was made in 1926 with the cooperation of the Bell Telephone Laboratories. The data obtained from this survey still are widely quoted. Because of the increase in population the then calculated 3,000,000 deafened school children in the United States is still near the truth, even though the 6 db. criterion for hard of hearing has been increased to 9 db.

their parents and schoolteachers and the general public in hygiene and common-sense measures for the prevention of deafness. These things it did to the best of its ability, as did all the organizations for the hard of hearing subsequently organized.[2]

During World War I the larger organizations grew to real professional status, and the recruiting officers for the armed services referred to them men rejected because of hearing defects. They arranged lip-reading scholarships and vocational readjustments for hard of hearing soldiers and sailors, and the experience gained proved invaluable a quarter of a century later in and after World War II. Now, 30-odd years later, several societies and some self-set-up "speech and hearing" centers and commercial "hearing aid clinics" are attempting to organize what they call similar services, thus far with but little success from the viewpoint of the hard of hearing. Some so-called "clinics" not sponsored or approved by the American Hearing Society seem more interested in selling hearing aids than in really helping the hard of hearing.

In 1919 Dr. Wendell C. Philips, then president of the New York League for the Hard of Hearing, brought the groups in the various cities together to form the American Federation of Organizations for the Hard of Hearing. (This name was a mouthful, or an earful; today it is a more tasty morsel, the American Hearing Society.)

Starting with perhaps only a place for meetings, many of the chapters soon moved to larger quarters, and some purchased quarters. With the advent of radio, in the early 1920's, headphone attachments were obtained for group teaching and group enjoyment. In 1920 free lip reading classes were instituted in the schools of some of the larger cities. In New York City alone over 11,000 requests were received from hard of hearing persons for advice and assistance in their problems, which varied from the selection of hearing aids to the advisability of undergoing finger inside the Eustachian tube manipulations, so-called "cures," magic ear oils, and fakes of various kinds. Several of the chapters even employed most efficient "Dorothy Dixes" to advise the lovelorn. Groups for the young and for old-timers were organized, and all made use of the recreational and social facilities. The chapters grew in size and in efficiency. Many exhibited several types of "ear phones." No charge was made for any of the services.

The chapters of the American Hearing Society throughout the country did more to popularize and expand the use of the hearing aid than any other agency, and the members and professional staffs took delight in wearing their aids so that all and sundry could see them and profit thereby. Many prominent people came to the league to help. I wonder whether the hearing-aid manufacturers understand and appreciate all this. Some of them criticize us because none of our organizations exhibit every type of hearing aid in their testing rooms. I ask them, "Does any manufacturer or dealer have every type of hearing aid in his salesrooms? Does he test people in his salesrooms with any aid except his own?" For obvious reasons the answer is, "He does not!" Many of our chapters, however, have exhibited as

2. I do not wish to overstress the New York League for the Hard of Hearing, but since I was active in it for over 30 years and its president for 8 years, it is difficult for me not to bring it frequently into the picture. Limitations in space and time prevent me from detailing the work of all our fine chapters and from giving the names of their valiant pioneers.

many hearing aids as have been approved by the American Medical Association Committee on Audiometers and Hearing Aids, a number greater than is necessary for a proper selection.

The organizations for the hard of hearing contributed much to the improvement of the odd-looking contraptions manufactured in the early days and to the reliability, standardization, and diminution in size of the batteries. In the beginning the hearing aid was only a carbon microphone, a telephone placed in or over the ear, with heavy dry batteries carried in a pocket clipped to the clothing or slung about the neck. This aid did magnify sound, but if it was not properly adjusted it made life unbearable by the loud "frying," "sizzling," and other noises it produced. Moreover, some of the original aids were as large as or larger than present-day television sets. An early so-called portable model weighed about 7 lb. (3.2 kg.). Nevertheless, the aids were avidly adopted by the different hard of hearing organizations for use in their speech-teaching, speech-reading, and speech-hearing clinics, for the rehabilitation and reeducation not of the hearing but of the hard of hearing person.

In 1926 the New York League for the Hard of Hearing established under my direction a clinic for the prevention of deafness at the Manhattan Eye, Ear and Throat Hospital. This clinic was the first of its kind and I believe set a pattern for the development of clinics in other boroughs and throughout the country. In this clinic every patient was given a complete otological examination, including audiometric testing in a sound-proofed room, stereoscopic x-rays of the petrous bone, differential blood determinations and blood chemistry studies, and in many instances basal metabolism determinations. Every item was listed and catalogued under the various categories of diagnosis. The records are still available. Today there are other clinics especially interested in prevention and rehabilitation, but without the assistance of properly functioning employment services and classes for children and their parents and for adults and the aged, they will not be able to do the work established and still being carried out by many chapters of the American Hearing Society. It is sometimes forgotten that the most valuable piece of clinical equipment is a trained and understanding operator, not a piece of electrical or mechanical apparatus.

In cooperation with Teachers College, Columbia University, the staff of Chapter 1, American Hearing Society, gave courses on the problems of the hard of hearing and on methods of teaching lip reading and speech training to deafened children. A textbook was prepared for teachers of the hard of hearing. All activities were under the supervision and active participation of Miss Estelle E. Samuelson, who has for many years been secretary of the New York League. Miss Samuelson wrote the first textbook for training of children, and Mrs. Ronnei wrote one on auditory training, "Learning to Look and Listen," and a book for both children and their parents, "Tim and His Hearing Aid."

During the "Hoover-Roosevelt depression era" there was widespread unemployment. The American Hearing Society, under the inspired guidance of its officers and loyal staff, headed by its executive secretary, our loved and lovely Betty Wright, valiantly persisted despite cuts in salaries and hardships among its workers and members. Jobs were obtained for hard of hearing workers. Many other welfare agencies failed to survive the depression, but no chapter of the American Hearing Society died. One of the most generous member contributors to the cause

was Mr. Starling W. Childs, who also was the largest contributor to the Central Bureau of Research, whose trustees are the custodians of the Research Fund of the American Otological Society, Inc.

The larger chapters set an example for the smaller organizations by establishing funds for supplying free hearing aids for the indigent and revolving funds for helping others to purchase aids. Many hearing-aid manufacturers sold us aids at cost and some from time to time donated aids. The American Hearing Society was the pioneer in setting up machinery for yearly examination and follow-up and auditory-training projects for children so that the residual hearing of even the severely deafened might be utilized to enable them to hold their own with normally hearing children in school, at play, and in the world. It is no longer unusual to see children happily wearing hearing aids. More should do so.

Nursery schools and classes for the instruction of parents were established and are now becoming widespread. Without parent cooperation in management and teaching, many deafened children flounder and become more and more emotionally unstable, as do their parents.

The American Hearing Society received national recognition and was asked to serve at the White House Conference on Child Health and Protection. The New York League prepared forms and cooperated with the federal government and the board of education in training the technical staff of the WPA project for testing the hearing of school children in the New York City schools. Chapters in other parts of the country rendered similar services.

In 1938, 1939, and 1940 several members of Chapter 1 served on a New York State legislative commission to study facilities for the hard of hearing. I believe this was the first commission of its kind in the country. In cooperation with the welfare council, information service was expanded and the New York League became a member of the Greater New York Community Chest. Other organizations of the American Hearing Society in increasing numbers have become members of the Community Chest in their areas. Under the presidency of Dr. C. Stewart Nash, the number of chapters participating increased to 50. Now I understand they number 70.

During the late 1930's and since, owing largely to World War II developments in electronics, hearing aids have been greatly improved and streamlined. The hearing aid and vocational-consultation services were swamped, and even today bookings are made six months or more in advance.

Before the Veterans Administration program was operative, a full-time service center was set up to meet the needs of returning war-deafened veterans of World War II. This was known as the "Veterans Sounding Post." It was established by the New York League in memory of the late Lieutenant Lester N. Hofheimer, United States Air Force, a casualty in India in 1943. It provided psychologic case work, home adjustment advice, lip-reading and speech instruction, hearing aid service, and recreational and employment services for all branches of the armed forces. The Caduceus Post 818, American Legion, has made yearly contributions to this activity.

The American Hearing Society is the voice of the hard of hearing people of America. It now has 117 chapters and over 10,000 members. (The goal should be over 100,000.) It has pioneered in all the services I have mentioned, and in

addition in organizing community-supported groups, in developing the screening phonograph audiometer technique, and in promoting state and national legislation for physically handicapped children, conservation-of-hearing programs in schools, National Hearing Week, and publication of a hearing journal, *Hearing News,* and hundreds of informative pamphlets.

Our statistics show that deafness affects more than twice as many persons as cancer, heart disease, tuberculosis, and infantile paralysis combined; yet fewer people join and still fewer contribute to organizations for the hard of hearing than join and contribute to agencies for these other conditions.

Although large numbers of social workers, school administrators, teachers, health and rehabilitation officers, and speech and hearing therapists are now among the members of the American Hearing Society in many locations, otologists in sufficient numbers have been conspicuous by their absence. This situation is unfortunate, because all organizations for the hard of hearing need their guidance. They need otologists who are cognizant not only of the diseases and disorders of the ear but of the social and psychological repercussions often involved in deafness. If otologists neglect this field of endeavor they will find that the so-called "audiologist," the "deaf cure," and "hearing restoration practitioners" will take over. It would almost seem that some otologists think that deafness is not a primary concern of otology. If correction and rehabilitation services are not problems of the otologist, then correction and rehabilitation are not problems of the ophthalmologists. The belief that less can be done for deafness than for blindness is a myth. In many instances much more can be done to improve the hearing than can be done to improve the eyesight. No eyeglasses can bring back the sight in cases of eye obstruction, but obstructive deafness is easily overcome by hearing aids and certain forms by operative procedures.

The hard of hearing are vitally concerned with the inheritance of deafness. Much specious preaching has been heard on this subject. It is not generally realized that human genetics is far from being an exact science, and therefore no one should try to play God. However, the chapters of the American Hearing Society have cooperated with the geneticists and otologists and have provided many cases for study in heredity. The majority of the identical twins under my observation for the past 10 years have been referred from two or three chapters of the American Hearing Society. We have been concerned with factors seemingly involved in the heredity of deafness and in the activation and exacerbation of the lesions of otosclerosis. Would that we could procure more voting men and women identical twins with family or individual histories of otosclerosis. The more we study the better will be our understanding of this great mystery in otology.

One of our problems has been unwarranted discrimination against the hard of hearing in industry and in certain professions. It required many years of persuasion to stop certain boards of education from dismissing competent teachers just because their threshold audiograms were not up to some arbitrary standard. In some instances the wearing of a hearing aid was used as an excuse for dismissal. Fortunately such practices are lessening. The scarcity of first-class teachers in our schools will help terminate them, because it will provide an opportunity for demonstration of how efficient the wearers of hearing aids can be.

Another problem is the medicolegal disability associated with hearing losses incurred in accidents or from exposure to industrial noises. This problem is acute. Solutions are being sought by several responsible committees. Recent technical advances make a satisfactory solution possible. The minimal requirements for speech audiometers as adopted by the Council on Physical Medicine and Rehabilitation of the American Medical Association have recently been released for publication. The academy committee and other responsible groups are favorably considering speech audiometry as a fundamental part of methods for assessing medicolegal disability. The time is ripe for standardization of speech audiometry for this purpose in correlation with other diagnostic and audiometric procedures.

Age has been used as a criterion of performance of the brains, the muscles, and the ears, but not so much of the eyes. It has recently been proposed that any one 50 years of age or older should suffer a deduction from his compensation for hearing loss, because on the average those over 50 do have some loss in high tones. There are so many variations and exceptions that such an arbitrary procedure is obviously wrong. What has the average got to do with it? Of interest is the individual performance, not an average at a given age. Do not let physicists and experimental psychologists tell physicians what their patient should hear. The ultimate criterion for evaluating hearing capacity is what the patient does hear, as determined by clinical observations and audiometric measurements checked with tests for speech hearing at different levels.

During the past 35 years there has been an increasing increment of understanding of the problems of deafness, of its etiology, pathology, detection, and alleviation. Many people have contributed and in many fields of endeavor, and it is not entirely fortuitous that these years have coincided with the growth and work of organizations for the hard of hearing.

Under the presidency of Dr. Thomas L. Tolan, the American Hearing Society is now stressing courses for training the executive directors and executive secretaries of chapters. Research projects and a cooperative plan for financing national and chapter organizations are in competent hands.

I close with an appeal to the otologists, the audiologists, the hearing-aid manufacturers and salesmen, and all good physicians and all good people everywhere: Do not belittle the American Hearing Society. Instead cooperate with it and help it wholeheartedly, because there is no better way to serve these courageous people, who have already helped so well themselves and the hard of hearing everywhere.

140 East 54th St.

OSTEITIS OF THE SPHENOID SINUS

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IN A PREVIOUS report [1] an alteration of the frontal sinus wall which occurred frequently in paranasal cancer was discussed. This was observed in 35% of our irradiated patients. The alteration appears on x-ray films as blurring, demineralization, and/or condensation of the upper and posterior wall of the sinus. It is not surprising to find similar alterations developing in the wall of the sphenoid sinus also.

MATERIAL

Our related experiences are limited for various reasons. The films of our irradiated patients were reviewed. In the early period the sphenoid sinuses were not examined routinely. Later the film technique was aimed at visualizing the soft tissue alterations rather than the finer bony details. Therefore, the greatest number of the lateral sinus films and a part of the axial views were not suitable for the present special study of the finer details of the sphenoid sinus outlines. It was especially difficult on many films to distinguish whether the observed thickening of the outline was due to that of the adjacent membrane or to condensation of the bony wall. Therefore, only a selected number of the films could be utilized for analysis. These were mainly axial views, and infrequently lateral films. The limitation to technically suitable, selected films makes this material insufficient for statistical conclusions, but suitable for a few general observations.

FREQUENCY

In spite of the fact that the frequency could not be established statistically, it was noted that the secondary alteration of the sphenoid sinus wall occurs not infrequently. It was found in 20 cases of the reviewed cancer material and in only 1 case of the current sinusitis material during the last year. In 12 of the 20 cancer patients the primary extensive disease was localized to the paranasal sinuses, in 3 to the oropharynx, and in 5 to the nasopharynx. The one sinusitis patient mentioned had a chronic pansinusitis of long duration, without malignant growth. While the equivalent alteration of the frontal sinus was observed in paranasal cancer, and less frequently in chronic obstructing sinus inflammations, the alteration of the sphenoid sinus wall was found also in cases of extensive cancer of the oropharynx and nasopharynx. This is explained by the different location of the sinus. Among the 12 cases

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1. Haas, L. L.: Secondary Alterations of the Frontal Sinus in Cancer of the Paranasal Sinuses, Am. J. Roentgenol. **66**:797-802, 1951.

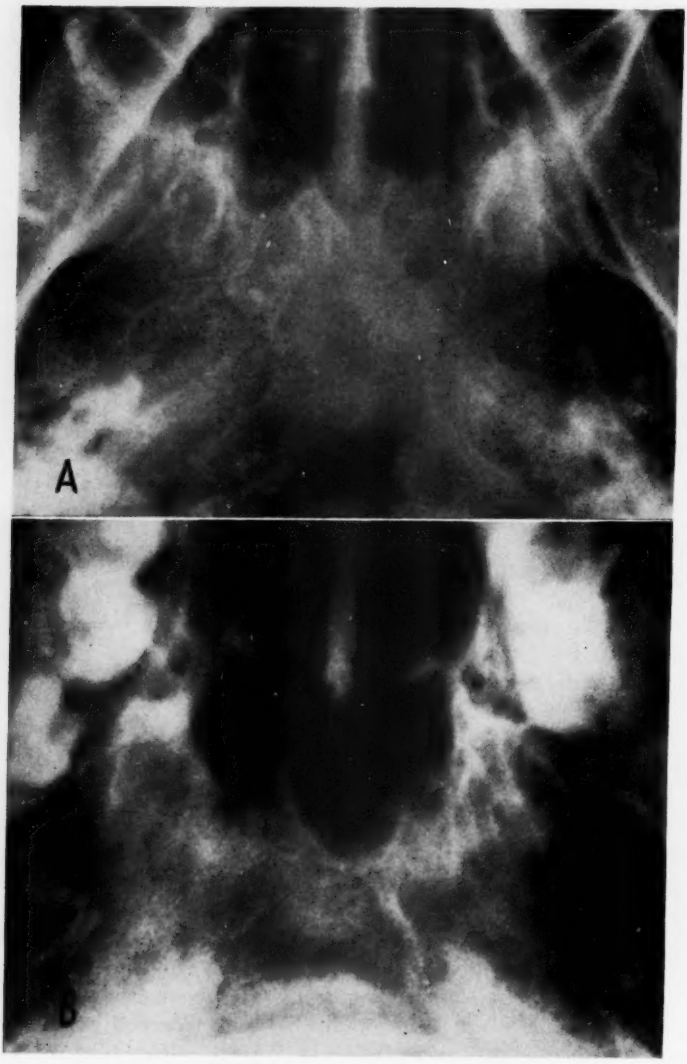

Fig. 1.—*A*, epidermoid carcinoma of the nasopharynx; demineralization, partial loss of the compact layer (white line) of the left sphenoid sinus wall. *B*, recurrent reticulum cell sarcoma of the nasopharynx; blurring, with loss of the white line of the right sphenoid sinus wall; slight condensation. *A* and *B* each indicates a separate case.

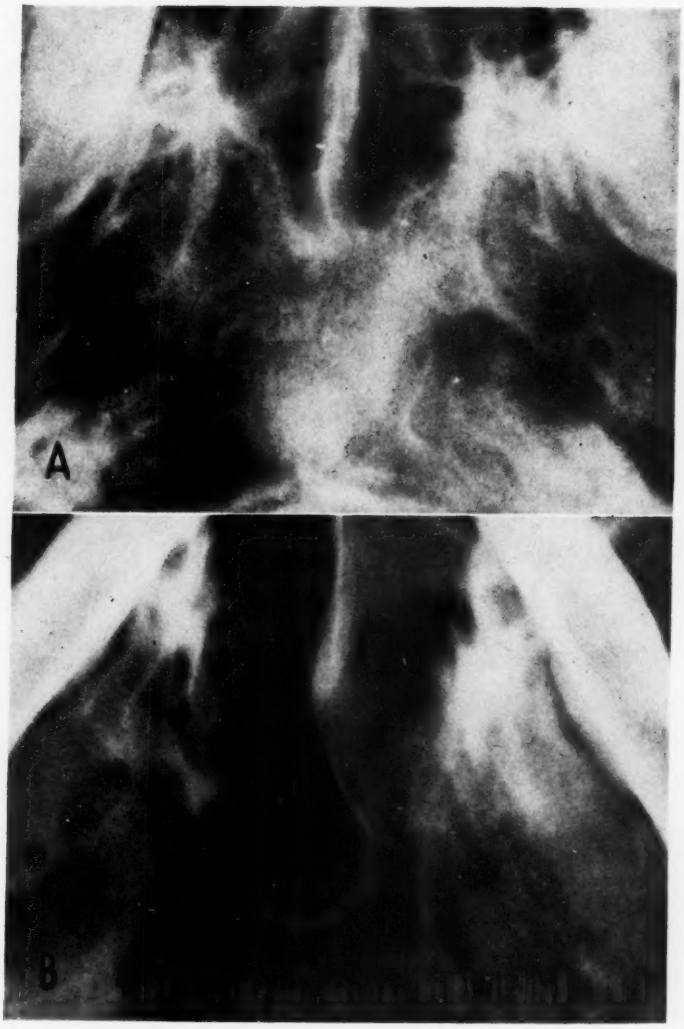

Fig. 2.—*A*, advanced epidermoid carcinoma of the oropharynx (tonsils, base of tongue) with extension into the nasopharynx; initial condensation of the right sphenoid wall, mainly in the posterior segment; tumor mass in the nasopharynx. *B*, papillary epidermoid carcinoma of the left nasal cavity, antrum, and ethmoids, extending into the nasopharynx; slight condensation of the right sphenoid wall in a small posterior segment. *A* and *B* each indicates a separate case.

of paranasal cancer with evidence of this alteration of the sphenoid wall, simultaneous alteration of the frontal sinus also was shown in 11 cases. Some of these antral cancers (e. g., Figs. 2*B* and 6) were very advanced, with extension into the nasopharynx. In some cases of oropharyngeal cancer (e. g., Fig. 2*A*) the lesion had already extended into the nasopharynx.

ROENTGENOLOGIC APPEARANCE

The roentgenologic appearance of the alteration is similar to that of the frontal sinus described in the previous report. One may occasionally find demineralization, with loss of the white line which represents the compact layer of the sinus wall (Fig. 1). The majority of the alterations appeared rather as a more or less marked

Fig. 3.—Epidermoid carcinoma of the oropharynx (left tonsil, anterior pilar, hard palate). Initial condensation of the left posterior sphenoid sinus wall.

condensation of the sinus outline. The thickening of the sinus contour or condensation of the surrounding bony structure is at times only slight and confined to small segments. Special attention may be needed to find it (Figs. 2 and 3). In other cases the condensation is conspicuous, more extensive, and more marked (Figs. 4 and 5). In Figure 6 it is very irregular. In some instances an even, narrow, white stripe is visualized, corresponding to a uniformly thickened sinus wall segment (Fig. 7). In Figure 8*A* nearly the entire course of the sphenoid sinus wall and septum is uniformly involved. The even thickening of the outlines is very striking in our single case of benign pansinusitis (Fig. 8*B*). Alterations similar to those shown in the last Figure were seen in the frontal sinus mainly in cases of benign inflammation. In frontal sinus alterations one could find differences of the details in cases of benign inflammation and in cases of cancer, respectively. In respect to the sphenoid wall alterations, our material was insufficient for such differentiating conclusions.

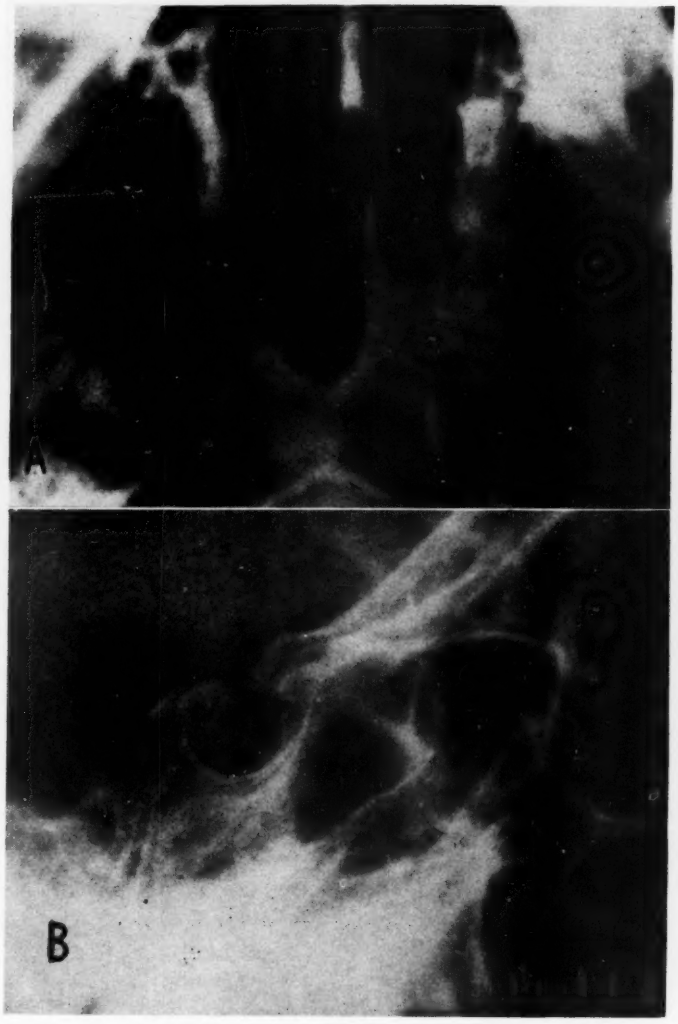

Fig. 4.—Epidermoid carcinoma of the left antrum, also involving the right antrum; originating from a cheek cancer. *A*, left sphenoid wall blurred, indistinct; adjacent soft tissue shadow within the sinus; white line of the right sphenoid sinus thickened, condensed. *B*, condensation of segments of the sphenoid sinus walls visible on the lateral view also.

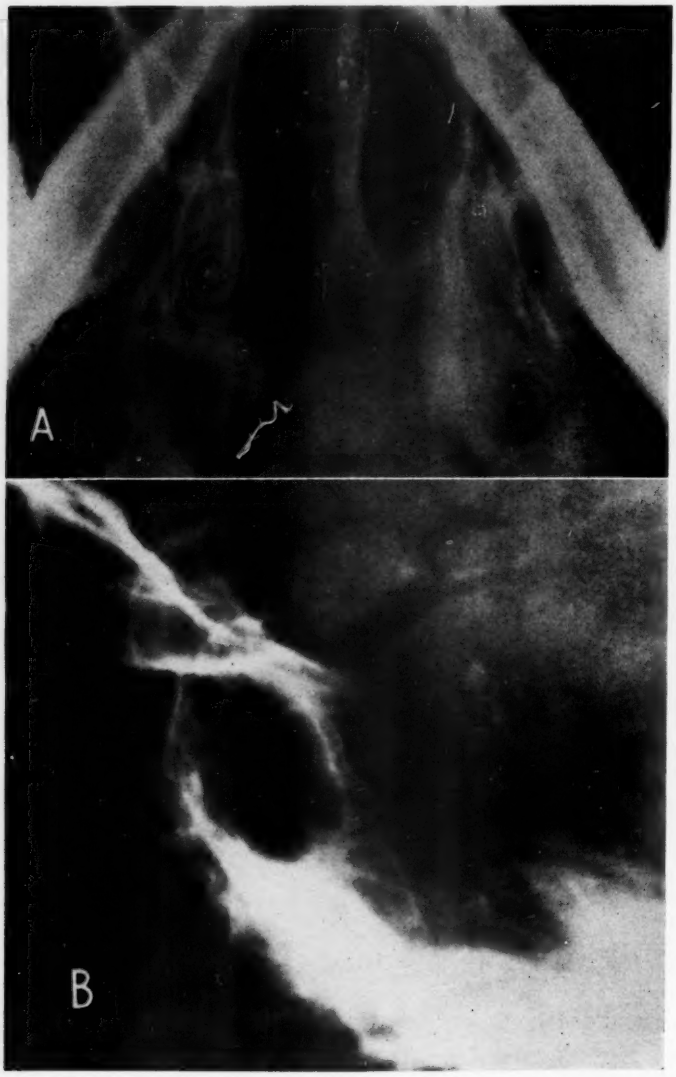

Fig. 5.—Advanced bilateral oropharyngeal carcinoma (base of tongue, pilars, pharyngeal wall, epiglottis). *A*, right sphenoid wall blurred, indistinct; left sphenoid wall condensed; otherwise intact white line; tumor mass in the left half of the nasopharynx; eroded left foramen ovale. *B*, lateral view also showing condensation of the posterior sphenoid sinus wall; demineralization of the sphenoid body and of the neighboring sella floor.

Fig. 6.—Squamous cell carcinoma of the left antrum, ethmoids, and nasal cavity, extending into the oropharynx, and nasopharynx, and the sphenoid sinus, with extensive destruction of the bony structures; marked condensation of bilateral sphenoid sinus walls.

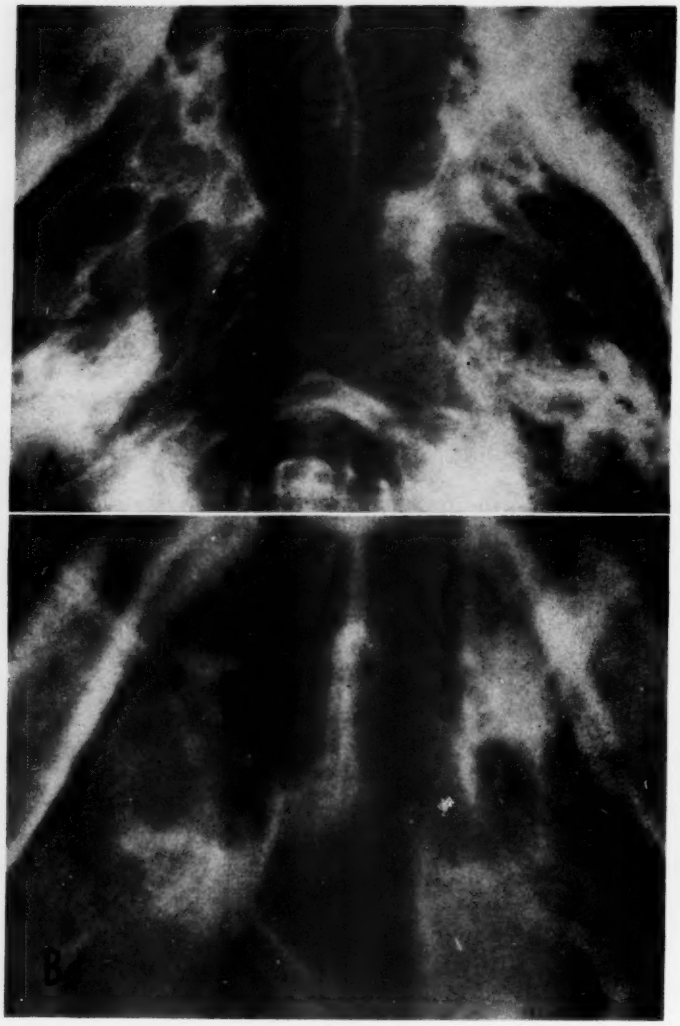

Fig. 7.—*A*, epidermoid carcinoma with sarcomatous qualities, involving the right antrum, nasal cavity, and hard palate; cerebral metastasis; narrow condensed stripe in the left posterior sphenoid wall. *B*, advanced epidermoid carcinoma of the right antrum, involving the ethmoids, canine fossa, hard palate; narrow stripe of condensation in the posterior walls of the lateral sphenoid sinus segments, mainly on the right side; right pterygoid plates destroyed. *A* and *B* each indicates a separate case.

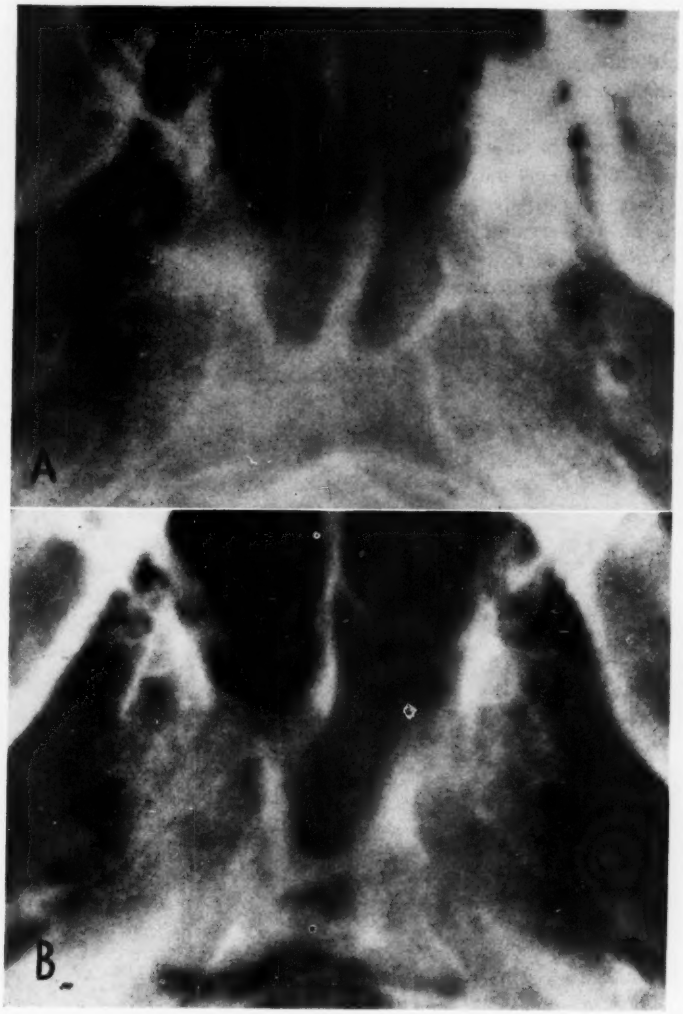

Fig. 8.—*A*, epidermoid carcinoma of the left antrum, with orbital invasion; postoperative recurrence; diffuse thickening, condensation of the bony walls of the sphenoid sinus and of the septum. *B*, bronchiectasis; chronic pansinusitis, mainly on the left; clinically not active; observed for over three years without roentgenological change; left sphenoid sinus cloudy; left sphenoid sinus wall and septum markedly thickened, condensed; right sphenoid wall demineralized. *A* and *B* each indicates a separate case.

PATHOLOGIC NATURE

The roentgenologic appearance of the alteration gives the impression of a low-grade secondary osteitis due to occlusion and infection caused by the tumor. The clinical observations also support this interpretation and contradict the explanation that this alteration of the sinus walls is due to secondary neoplastic involvement. On one hand, one rarely finds progression of the secondary alteration, and under such circumstances only at a very slow rate during longer periods of observation, while during the same time the neoplastic bony destruction rapidly progresses. On the other hand, patients may remain asymptomatic a longer time after surgery or irradiation, in spite of the fact that these secondary sinus wall alterations were untreated. This observation time in our material extends up to three years. The difficulties of direct microscopic proof by biopsy or necropsy were mentioned in the paper on the frontal sinus alteration and could not be overcome until now.

COMMENT

The presence of these secondary changes of the sphenoid sinus wall calls for careful clinical and x-ray examination for diagnosis or exclusion of paranasal, nasopharyngeal, and oropharyngeal cancer, and for differentiation from chronic obliterating sinus infection.

NEW PNEUMOPHONE

An Economical and Simple Diagnostic Instrument for Rapid Quantitative Determination of Eustachian Tubal Function

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ELGIN, ILL.

THE PRINCIPLE of listening for a change in sound intensity with change in tension of the tympanic membrane has been known for more than 70 years, since the writing of Lucas and Gellé, and is now fully accepted by recent researchers in the field.[1] Taking cognizance of the clinical importance of this technical principle, van Dishoeck in 1937 introduced a practical pneumophone for accurately evaluating Eustachian tube function.[2] No doubt because of the complexity and the cost of the pneumophones used by the researchers, there has been very little appeal to the practicing otolaryngologists to use them in routine office practice. To overcome these disadvantages, I am presenting a new apparatus.

Van Dishoeck [3] stated that a very simple pneumophone could be constructed by fixing the bone receiver of an audiometer to the outer wall of a small metal chamber provided with an earpiece. The 500 cps tone of the receiver is transmitted through the wall to the air in the chamber and the external meatus, the pressure in the chamber being changed in the same manner as in an ordinary pneumophone. I have further simplified this by using the end of a diaphragm-type stethoscope,[4] laying it on top of the air conduction earphone of an audiometer, securing it with a rubber band or sponge rubber cuff,[5] and, by means of plain glass T-tubes and rubber tubing, connecting it up to an ordinary water manometer, which is also readily commercially available at very small cost.[6] The earpiece is made of a 3 in. (7.6 cm.)

1. van Dishoeck, H. A. E.: Tubal Disorders and the Pneumophone, Acta oto-laryng. **41**: 196-203, 1952. Perlman, H. B.: Quantitative Tubal Function, Arch. Otolaryng. **38**:453-465, 1943. Wever, E. G.; Bray, C. W., and Lawrence, M.: The Effects of Pressure in the Middle Ear, J. Exper. Psychol. **30**:40-52, 1942. Wever, E. G.; Lawrence, M., and Smith, K. R.: The Effects of Negative Air Pressure in the Middle Ear, Ann. Otol. Rhin. & Laryng. **57**:418-428, 1948. Rasmussen, H.: Studies on the Effect on the Air Conduction and Bone Conduction from Changes in the Meatal Pressure in Normal Subjects and Otosclerotic Patients, Acta oto-laryng. Supp. 74, pp. 54-64, 1948. Jones, M. F., and Edmonds, F. C., Jr.: Acoustic and Vestibular Barometry, Ann. Otol. Rhin. & Laryng. **58**:323-344, 1949. Rudmose, H. W.; Clark, K. C.; Carlson, F. D.; Eisenstein, J. C., and Walker, R. A.: The Effect of High Altitude on the Threshold of Hearing, J Acoust. Soc. Amer. **20**:766-770, 1948.

2. van Dishoeck, H. A. E.: Das Pneumophon: Ein Apparat zur Druckbestimmung im Mittelohr, Arch. Ohren-, Nasen- u. Kehlkopfh. **144**:53-58, 1937.

3. van Dishoeck,[1] p. 198.

4. Pilling Special Stethoscope, Bowle's Patent, George P. Pilling & Sons Co., Philadelphia.

5. The Maico Company, Inc., Minneapolis.

6. E. H. Sargent & Co., Chicago.

piece of glass tubing, on the end of which is fitted a 1 in. (2.5 cm.) piece of ½ in. (1.3 cm.) soft rubber tubing, making an airtight coupling to the external ear canal when it is applied with firm pressure by the patient. This modified apparatus has produced very satisfactory results for all practical purposes, is economical, and can be assembled in a minute or so.

METHOD OF OPERATION

It should be understood that there is a transmission loss of about 20 db. because of the apparatus. However, this can be readily compensated for by turning up the audiometer intensity the required amount. Also, it has been found that the testing should be done with a sound intensity about 20 to 30 db. above the threshold,

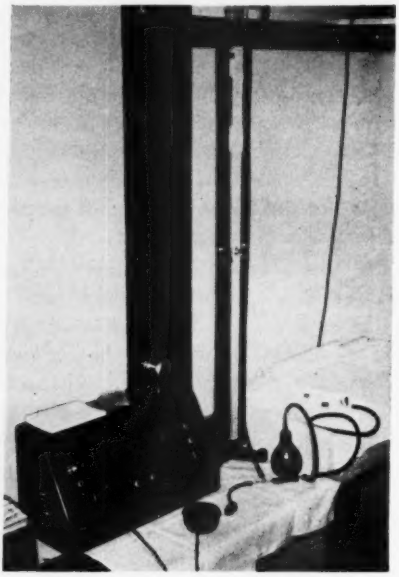

Fig. 1.—Pneumophone. Note the audiometer, air-conduction earphone, diaphragm bell type of stethoscope, and earpiece connected by rubber tubing and glass T-tubes to the water manometer.

because the loudness change can be recognized more easily at such a level than at threshold. Thus, in routine testing with the pneumophone the air conduction earphone is usually set at 40 to 50 db. above threshold in order to make up for the above-mentioned losses.

By keeping the following points in mind, the required data are procured from the patient with a minimum of effort and in less than a minute.

1. The patient must be shown how to hold the earpiece gently but with firmness snugly in the orifice of the external canal so that no air will escape when positive or negative pressure is made with the Politzer bag.

2. The operator uses digital pressure of one hand on the ball valve of the bag; with the other hand gently squeezing the bag, alternating pressure can be obtained by proper coordination, thereby raising or lowering the column of water centimeter by centimeter.

3. The patient is instructed to watch the column of water and while listening to the 500 cps. sound to point out the level at which the sound intensity reaches a maximum. Almost everyone is easily able to mark this point, within 2 or 3 cm. of water, consistently every time. A value about 20 or 30 db. above threshold is used for optimum results.

4. It has been noted that in older patients there is a tendency for the external canals to close off; thus the sound suddenly stops because the meatal rim of the conchal cartilage pushes forward when the earpiece is secured firmly to the canal. This mechanical difficulty is readily overcome by the preliminary insertion of a ½ in. piece of firm rubber tubing into the outer third of the canal with bayonet forceps. This tubing should be about ¼ in. (0.6 cm.) in outer diameter and within a lumen of about ⅛ in. (0.3 cm.); such tubing may be procured from any cystoscope instrument supply house.

It is believed that the qualitative tests of tubal function are not so infallible as heretofore supposed. Although their correct use should not be discounted, such as in auscultation, for instance, with tubal inflation and differentiation whether it is a

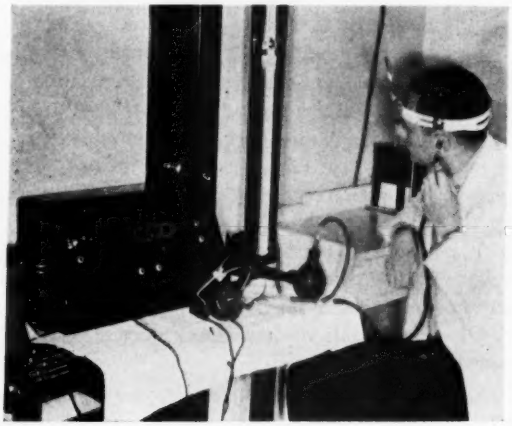

Fig. 2.—Apparatus in use. The earpiece is held in the meatus by the patient; the bell of the stethoscope has been tucked under the collar of the air-conduction earphone to pick up the sound from the audiometer.

"phut" or a "whoof" one hears, too great reliance is placed on clinical judgment in introducing the correct amount of inflationary air pressure to produce the sound.[7] The amount of pressure in itself is a most illusory quantity.

CLINICAL APPLICATIONS OF THE PNEUMOPHONE

1. *Differential Diagnosis Between Middle Ear and Labyrinthine Pathology.*—Although the qualitative tests of tubal function are valuable for the gross determination of tubal patency, it is much more revealing and less subjective to get quantitative readings of scientific accuracy. In my opinion, it is quite possible to have a case in which dry tubal catarrh manifests itself in symptoms of extreme vertigo, tinnitus, and deafness, thus masquerading as Ménière's syndrome, which it would be possible to treat with nicotinic acid, low salt diet, etc., when in reality other therapy is required. On the other hand, it is quite possible to find cases of automatic dysfunction mistaken for tubal catarrh because the patient had stated that politzeri-

7. Hoople, G. D.: Otitis Media with Effusion: A Challenge to Otolaryngology, Laryngoscope **60**:315-329, 1950.

zation gave temporary relief. In my own clinical experience with the use of the pneumophone, more and more cases of labyrinthine dysfunction are being diagnosed which had heretofore been labeled as Eustachian catarrh.

2. *Serous Otitis Media, Eustachian Catarrh, and Tubal Stenosis.*—There are certain pathological states which must be appraised with caution when interpreting the pneumophone tests. It is my opinion that in the serous type of otitis media of the early stages when the tympanic cavity is nearly full of fluid there will be little change in intensity of the sound when alternating positive and negative pressure is made with the bag, though it will be noted that the threshold will be raised as indeed it is in otosclerosis very similarly. However, as the condition progresses from day to day there is less and less fluid in the middle ear, and the greater the air-containing cavity—but at a reduced atmospheric pressure—the greater will be the reading in centimeters of water on the manometer. Finally, as the tubal obstruction is overcome in the last stages of the catarrh, the built-up negative pressure will rather quickly entirely disappear at the end of several weeks. The test becomes a very valuable prognostic sign, and after return to normal function of the Eustachian tube one may with confidence say that the disease has been cured.

On the other hand, in the dry type of tubal catarrh with tubal stenosis there is always a marked negative tension on the tympanic membrane, except when the drum membrane is plastered up against the medial wall and no air is present in the middle ear. Every patient who is seen repeatedly with a negative tension of more than several centimeters of water and whose negative tension suddenly disappears after tubal inflation, with concomitant temporary increase in auditory acuity, week after week should obviously be given a diagnosis of chronic Eustachian stenosis. In addition, a patient may have a negative reading for a few times as a result of a recent upper respiratory infection, but after a few weeks this will usually disappear if there is no real chronic stenosis present.

3. *Routine for Aviation Personnel, Caisson Workers, and Airplane Passengers.*—All cases involving aviation personnel should be routinely studied by recorded quantitative readings. The incidence of barotrauma is so common that the pneumophone provides the test *par excellence* for the scientifically correct appraisal of tubal function in addition to routine nasopharyngoscopic examination. No doubt many latent cases will readily be discovered prior to exposure to changes in atmospheric pressure. Certain business executives who do much flying will do well to have pneumophonic examinations. It must be strongly emphasized that the difficulties hardly ever arise from decompression but are due rather to compression; it is when the air pressure in the middle ear becomes less than the surroundings and swallowing or chewing movements fail to equalize the pressures that pathological changes ensue.

4. *Surveys on Effects of Radium Therapy.*—It is logical to assume that all scientific surveys of the use of radium or other radiation therapy for the correction of tubal function should be checked by simple determinations of this type, which take so little time to perform. A more accurate measuring stick could scarcely be thought of.

5. *Routine Testing in Cases of Otosclerosis.*—There are two different tests which may be done with the pneumophone. Besides measuring the amount of tension in centimeters of water on the tympanic membrane which results in the most

efficient transmission of sound into the cochlea, i. e., the place on the manometer at which the 512 cps frequency pure tone is a maximum, it is also very simple to measure the sensitivity of each drum membrane and ossicular chain to tension change, as in the Gellé test. Accurate threshold values should be arrived at, necessitating whole new control figures, which as yet are only approximately given by van Dishoeck, who says:[8]

> In extreme cases of Otosclerosis, sound transmission being very poor, no deadening of the sound on changing the pressure will be noted, viz., the test of Gellé is negative. At the onset of this disease, the stiffness of the ossicular chain is already increased by the fixation of the stapes. Thus, the stiffness produced by the pressure on the eardrum will be of lesser effect. This can be measured by establishing the hearing loss produced with a 50 cm. pressure. Normally this loss is about 20 Dbl. In Otosclerosis it will be less depending on the degree of fixation of the stapes.

SUMMARY

A simple and economical pneumophone is described, and directions for its operation are given. Some of the commoner indications for its use are discussed, as in differential diagnosis between labyrinthine pathology and middle ear conditions; uses in serous otitis media, Eustachian catarrh, and tubal stenosis; as a routine examination for aviation or caisson worker personnel; in scientific surveys dealing with irradiation of the tube, and as a routine in testing of patients with otosclerosis for fenestration.

This pneumophone puts into the hands of every practicing otolaryngologist a diagnostic instrument as important and as practical as the stethoscope is to the general practitioner. Henceforth, the medical researcher into Eustachian tube pathology should include it among his equipment for the accumulation of scientific data as important as the audiogram itself.

164 Division St.

8. van Dishoeck,[1] p. 203.

Clinical Notes, New Instruments and Techniques

DIAGNOSIS OF TUMORS OF THE NASOPHARYNX BY CYTOLOGICAL PROCEDURE

Preliminary Report of a New Technique Employing a Suction Cannula

SIDNEY RUBENFELD, M.D.
AND
RUTH WINSTON, M.S.
NEW YORK

This is a preliminary report describing a simple cytological procedure in which a suction cannula was employed to establish a diagnosis of carcinoma of the nasopharynx. The suction cannula, to obtain cells for cytologic study, has found its greatest application in gynecological tumors. The simplicity of the aspiration technique prompted its trial for use in tumors of the nasopharynx.

The epithelium of the nose and throat has been made the subject of cytological study notably described in three recent reports. Morrison, Hopp, and Wu [1] examined smears from 85 patients and found malignant cells in 7. Fitz-Hugh, Moon, and Lupton [2] isolated 6 instances of malignant cells in antral washings from 72 patients. Friedmann,[3] reporting from London, isolated malignant cells from 105 patients who were suspected of having malignant tumors. He employed ordinary cotton swabs for the nose and curved wool carriers for smears from the nasopharynx.

TECHNIQUE

With the patient in sitting position, the nasal cavity is cocainized with cotton swabs in the usual fashion. A metal cannula (Fig. 1) is inserted along the floor until it strikes the posterior pharyngeal wall. A 50 cc. syringe attached to one end of the cannula creates the necessary suction. While the vacuum is maintained, the tip of the cannula is moved in and out to touch the mucosal wall at alternate movements. The pressure traumatizes the membrane and sucks in small fragments of tissue. The cannula and syringe are removed, and upon subsequent filling of the syringe with air the contents of the cannula and syringe are forcefully squirted onto glass slides. The material is spread as thin as possible and immediately immersed in fixative. The fixative in this study was composed of 75% tertiary butyl alcohol and 25% ethyl phosphate. Its use was described by Davidson, Clyman, and Winston in 1949.[4]

Associate in Radiation Therapy, Hospital for Joint Diseases; Associate Professor of Clinical Radiology at New York University-Bellevue Medical Center (Dr. Rubenfeld); Research Cytologist, Hospital for Joint Diseases (Mrs. Winston).

This work is being done at the Hospital for Joint Diseases, New York, as a combined effort of the Radiation Therapy Department and the Cancer Prevention and Detection Center, under special grant from the League for Cancer Research.

1. Morrison, L. F.; Hopp, E. S., and Wu, R.: Diagnosis of Malignancy of Nasopharynx: Cytological Studies by Smear Technic, Ann. Otol. Rhin. & Laryng. **58**:18, 1949.

2. Fitz-Hugh, G. S.; Moon, C. N., and Lupton, C. H.: Cytological Smear Technique in Diagnosis of Carcinoma of Maxillary Sinus: Preliminary Report, Laryngoscope **60**:376, 1950.

3. Friedmann, I.: Exfoliative Cytology as Aid in Diagnosis of Tumours of Throat, Nose, and Ear, J. Laryng. & Otol. **65**:1, 1951.

4. Davidson, H. B.; Clyman, M., and Winston, R.: New Fixative for Vaginal Smears, Stain Technol. **24**:145, 1949.

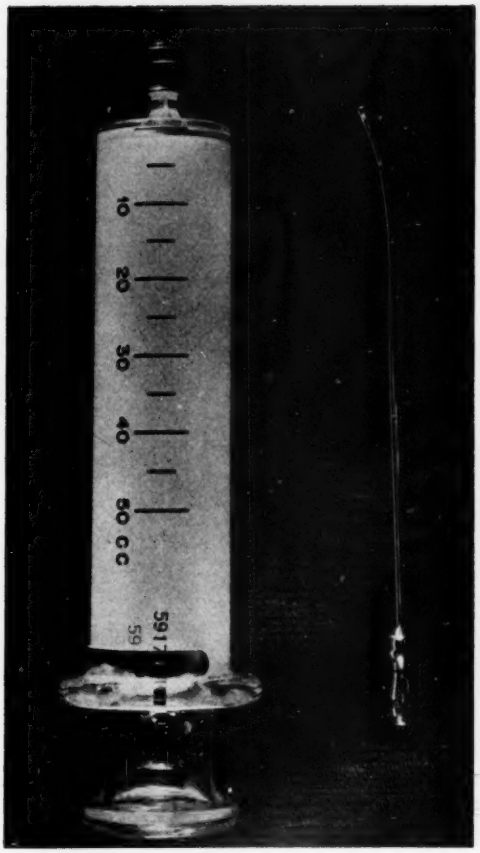

Fig. 1.—A thin metal flexible cannula is used. An ordinary 50 cc. glass syringe creates the necessary suction to aspirate the cells.

Fig. 2.—Cells aspirated from a normal (without tumor) nasopharynx; a cluster of ciliated columnar cells showing isonucleosis and isocytosis. Note the homogeneous distribution of the chromatin and the ciliated cytoplasmic proliferations.

Fig. 3.—Small clump of cells from a normal nasopharynx, showing squamous epithelial cells with normal nuclear-cytoplasmic distribution.

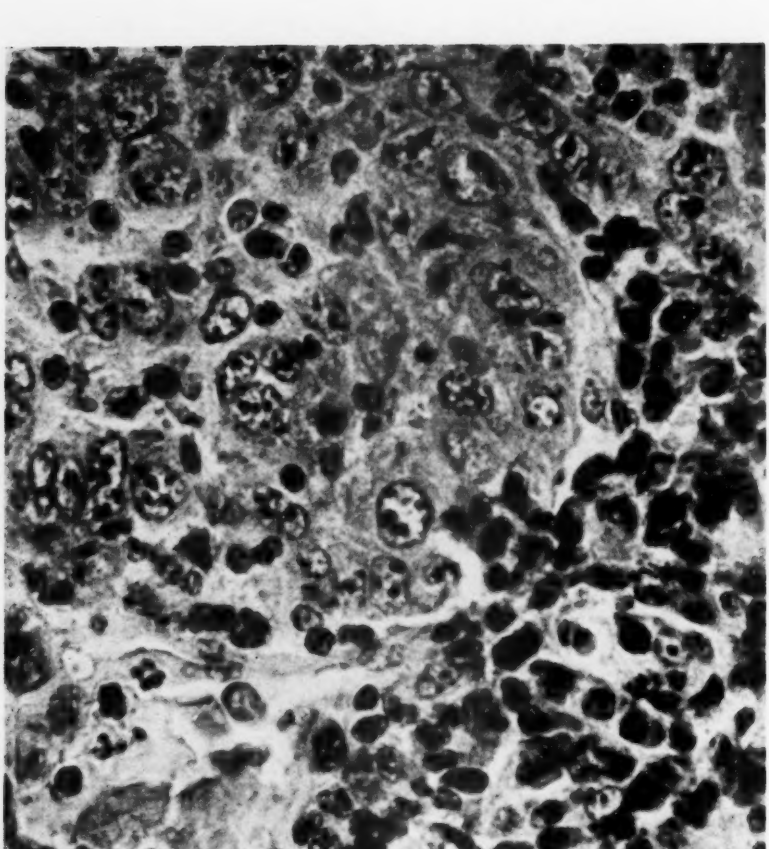

Fig. 4 (Case 1).—Photomicrograph of section of cervical node. The node structure was completely replaced by sheets of large, clear epithelial cells with distinct nucleoli. Note the strand of lymphocytes and plasma cells.

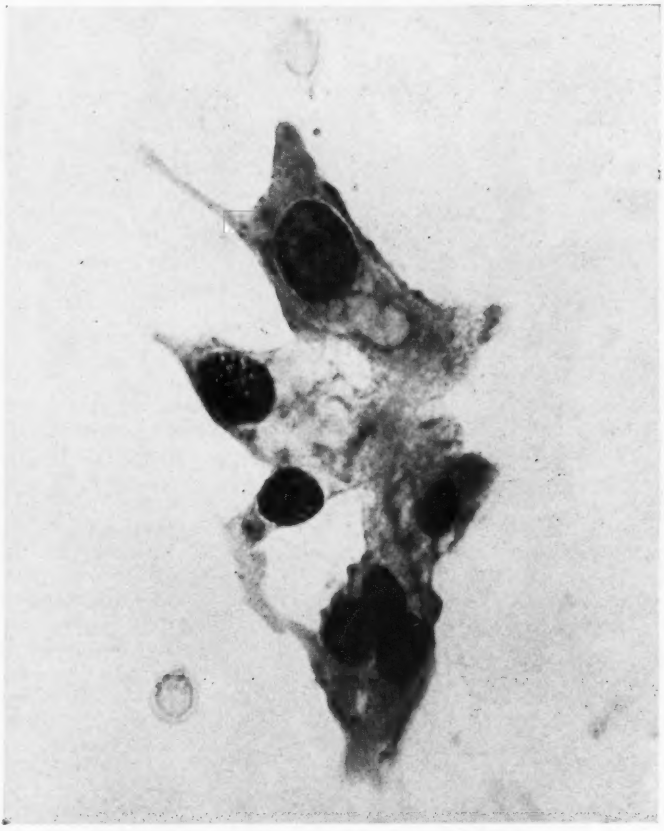

Fig. 5 (Case 1).—High power photomicrograph of cells obtained by aspiration showing a cluster of neoplastic squamous epithelial cells with irregular clumping of chromatin distribution, hyperchromatic nuclei, and vacuolation within the cytoplasm. The cells show anisocytosis.

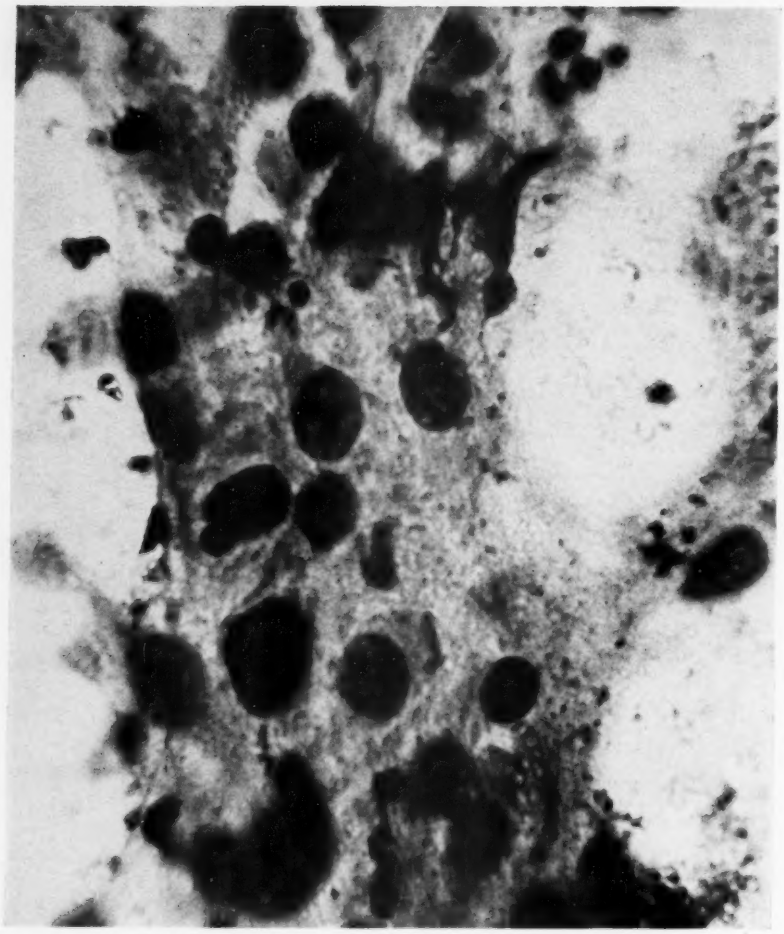

Fig. 6 (Case 2).—High power photomicrograph of aspirated cells, revealing a group of malignant squamous epithelial cells, the nuclei of which show irregular chromatin clumping, hyperchromatism, and anisonucleosis.

FINDINGS

In order to establish the normal cytological pattern, nasopharyngeal material was aspirated from 15 patients without head and neck tumors, so-called normals. The normal nasopharynx is lined with epithelium varying in nature from a stratified nonkeratinized type to a ciliated pseudostratified columnar cell. The cytologic pattern of these cells is depicted in Figure 2, in which are seen ciliated columnar epithelial cells showing isonucleosis and isocytosis. The chromatin content within the nucleus is homogeneously distributed. The ciliated cytoplasmic proliferations are clearly distinguished. Other smears, as in Figure 3, reveal clusters of squamous epithelial cells with normal nuclear-cytoplasmic distribution.

To illustrate alterations from the normal, the cases of two representative patients in whom cytologic criteria of malignant tumor were found are presented.

CASE 1.—A 57-year-old Chinese man was seen in November, 1951, because of generalized skin eruption of four months' duration. A diagnosis of dermatomycosis was made. During the course of routine physical examination, a group of lymph nodes along the left jugular and spinal accessory chains was found. The nodes varied from 2 to 4 cm. in length and were discrete, nontender, and freely movable. No other nodes were felt. The liver and spleen were not palpable. Findings on the remainder of the physical examination, including a neurological examination, were normal. Routine laboratory studies gave complete normal values. X-ray examination of the skull and accessory sinuses disclosed thickened mucosa of the left antrum. Punch biopsy of a mound of tissue from the vault of the nasopharynx was interpreted as nonmalignant. Thereafter one node in the left jugular chain was biopsied. Histological examination (Fig. 4) showed the lymph node structure to have been replaced by sheets of large, clear epithelial cells with distinct large nucleoli. Mitoses were common. A diagnosis of lymphoepithelioma in the lymph node was made.

At this time, a smear of the nasal cavity was taken in the manner described above (this case was observed at Bellevue Hospital). Study of these smears revealed numerous islands of cells, which presented variations in size and shape. Irregularity in chromatin arrangement was a conspicuous feature; cytoplasmic vacuoles were seen. These characteristics justified the diagnosis of malignant tumor in the nasopharynx (Fig. 5).

CASE 2.—A 20-year-old Negro man had a swelling of the right side of the neck for 10 days and a right temporal headache for six months. On examination, there was found a group of lymph nodes occupying the entire right jugular chain, forming a firm, nonfixed mass. Direct examination of the nasopharynx uncovered a mushy, friable, grayish tumor which depressed the soft palate on the right side. A biopsy specimen taken from the mass showed large, clear, vesicular malignant epithelium cells accompanied by groups of lymphocytes. A diagnosis of lymphoepithelioma was established.

Early in the course of x-ray treatment, cells from the nasopharynx were aspirated with the suction cannula utilizing the simple procedure outlined above. This was done to corroborate the malignant cytological picture obtained in other patients. A photomicrograph of one smear is shown in Figure 6. The cells were irregular in size, shape, and staining, most being hyperchromatic. The chromatin was irregularly clumped. Several cells show cytoplasmic vacuolation. These were malignant cells.

COMMENT

This suction cannula technique may be useful in searching for cryptic tumors in the nasopharynx. Its simplicity makes it useful (1) when a biopsy specimen is unobtainable, owing to the size of the growth (as in the nasopharynx); (2) when a patient refuses consent for a biopsy; (3) in the presence of palpable lymph nodes when a biopsy shows no conclusive evidence of a tumor (as in Case 1); (4) to follow the effects of treatment, and (5) to establish the diagnosis of recurrences after treatment.

SUMMARY

1. A simple suction cannula technique was used successfully to obtain cells from the nasopharynx for cytologic study.

2. The cytologic pattern in the normal mucosa is illustrated.

3. Two instances of malignant tumor seen in aspirated cells are briefly presented.

755 Park Ave.

POSTSUBMUCOUS TUBE PACK AND FREE BREATHING

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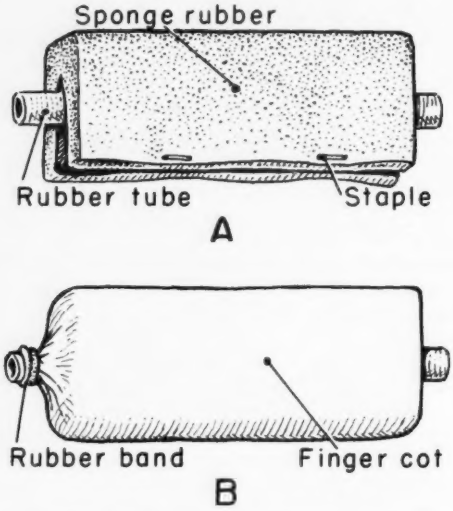
NEW YORK

Many patients will not undergo septal surgery because of their fear of complete nasal obstruction due to packing for the first 24 hours after operation. I was therefore moved to devise a packing through which a patient could breathe in comparative comfort after intranasal surgery.

The materials needed for the construction of this pack are a piece of soft sponge rubber approximately 1½ to 2 in. (3.8 to 5 cm.) long, ½ to ¾ in. (1.3 to 1.9 cm.) wide, and ½ in. thick. In addition to the sponge rubber, a soft ¼ in. (0.6 cm.) rubber tube, a finger cot, and two small wire clips are required. The measurements vary according to the size of the nose to be packed.



Tube pack to be used after intranasal operation.

To construct the pack, a cut is made along the side of the sponge rubber over its full length and is carried through about three-fourths of its width. The tube is inserted between the flaps thus formed and is long enough to protrude ¼ in. from each end. The wire clips are used to fasten the edges of the rubber flaps together in such a way as to enclose the tube firmly. The rubber finger cot is then drawn over the sponge-rubber tube pack. A small hole is made in the end of the finger cot, the tube being allowed to protrude a short distance beyond it. The cut edges fit tightly about the tube. The other end of the finger cot is fastened with a rubber band. The rubber band should be snug enough to keep secretions out of the sponge rubber, without compressing the airway. The rubber tube and the free edges of the finger cot are cut away evenly.

After the septal resection, a flat smooth tube pack, of the proper size and consistency, is lubricated with KY jelly and is inserted into each side of the nose. If more pressure is desired to prevent hematoma of the septum, a supplementary pack consisting of a finger cot filled

lightly with ½ in. dry gauze or a 1 in. sterile bandage is fitted in above the tube pack on the left side. I use a secondary pack on this side because this side is more often traumatized from incision and laceration.

When inserting the tube pack into the left nostril, I always use a flat, thin, wide retractor or long-bladed nasal speculum to cover the sometimes lacerated mucosa, in order to prevent the mucosa from being pushed posteriorly and thus exposing the right mucosal flap. Such an accident may result in a wide perforation, especially if there is the slightest laceration of the right mucous membrane, like that which may follow removal of a sharp spur. Great care must also be taken that no more pressure than is safe be exerted by the packs against the septal flaps.

To sterilize the pack, immersion in an alcohol bath for the usual period is all that is required, and any seepage into the sponge rubber can easily be expressed by pressure.

The intranasal tube pack suggested is smooth and moist, producing uniform pressure against the septal flaps. Its removal is not accompanied by bleeding because of the smooth rubber covering and because of the uniform pressure exerted on the septal membranes. The pack may be adjusted as to length, width, and thickness to the individual need. The tube is firm enough to remain patent and touches no mucous membrane at any time, because the ends do not protrude from the rubber sponge.

Occasionally the tube becomes filled with blood or postnasal secretions. The secretions can be aspirated or blown back into the posterior part of the pharynx with a 5 or 10 cc. syringe by the patient or the physician.

After 24 hours, all packs are slowly removed, without pain or bleeding. The patient has been comfortable and has been able to sleep and breathe freely throughout the crucial first 24 hours after the operation.

103 E. 78th St.

Case Reports

SCLEROMA IN PANAMA

Report of Two Cases

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PHILADELPHIA

THE REPORT of Reyes [1] indicates that scleroma is commonly observed in El Salvador and Guatemala. Since the climate, economic factors, and racial groups found in Panama are similar to those of these countries, one would assume that this disease should be observed with the same frequency in Panama. A check of the files of local hospitals revealed only one case in addition to the two cases to be described. The following are reports of two patients treated by the Ear, Nose and Throat Service of Gorgas Hospital.

V. C., a 25-year-old white man, born in Salvador, where he resided until 1941, was admitted to the Gorgas Hospital in March, 1949, for diagnosis and treatment of granulations of the anterior nares. The patient's chief complaints were partial nasal obstruction and hoarseness of several years' duration. Examination revealed the anterior nares to be occluded by granulations. The posterior surface of the soft palate, the arytenoids, and the subglottic area presented similar lesions.

Biopsy of material from the nasal lesions was reported as follows: "The microscopic examination reveals a thin layer of squamous epithelium showing a mild parakeratosis. In the subjacent tissue, there is a dense cellular infiltrate which is composed chiefly of plasma cells. Russell bodies are scattered throughout the section. There are clusters of large foamy cells with central nuclei; these are prominent. Small foci of neutrophiles and an occasional eosinophile are seen. Sections stained with silver by Steiner's method reveal large ovoid intra- and extracellular bodies morphologically typical of Klebsiella rhinoscleromatis." Cultures from this area were reported positive for Klebsiella rhinoscleromatis. Treatment consisted of 28 gm. of streptomycin administered in 0.5-gm. doses every six hours.[2] Upon completion of treatment the only residual pathology was scarification of the anterior nares, with reduction of the lumen to 3 to 4 mm. in diameter.

One month later the patient was treated for an acute suppurative sinusitis, at which time an additional 32 gm. of streptomycin was given. Cultures at this time were reported negative for Klebsiella rhinoscleromatis.

The patient was seen at irregular intervals up to September, 1951, when a granulation of the right naris was noted. Biopsy and culture confirmed recurrence of the original lesion. At this time a third course of streptomycin, consisting of 42 gm., was administered. Again cultures became negative and the lesion disappeared. The patient was last examined in June, 1952, at which time there was no sign of activity present.

1. Reyes, E.: Rhinoscleroma: Observations Based on a Study of 200 Cases, Arch. Dermat. & Syph. **54**:531-537 (Nov.) 1946.

2. (*a*) Hara, H. J.; Pratt, O. B.; Levine, M. G., and Hoyt, R. E.: Scleroma, a Clinico-Pathological Study of Seven Cases in One Family, Ann. Otol. Rhin. & Laryng. **56**:769-783 (Sept.) 1947. (*b*) Levine, M. G., and Hoyt, R. E.: Diagnosis of Scleroma, Arch. Otolaryng. **47**:438-445 (April) 1948. (*c*) Morwitz, S. M., and Horwitz, I. D.: Rhinoscleroma: Report of a Case Treated with Dihydrostreptomycin, Ann. Otol. Rhin. & Laryng. **59**:569-571 (June) 1950. (*d*) Ćurković, M.: Streptomycin in Treatment of Scleroma: Report of 3 Cases with Successful Results, Arch. Otolaryng. **52**:253-256 (Aug.) 1950.

R. G., a 21-year-old man born in Panama City, where he has resided up to the present time, was referred to the Out-Patient Clinic in May, 1951. His complaint was that of nasal obstruction of nine months' duration, with occasional serosanguineous drainage from the nose. Five years previously the patient had similar symptoms, which disappeared without treatment. He was not seen by a physician at that time. Examination revealed granulations extending from the anterior nares posteriorly to the soft palate. The anterior nares were almost occluded. Pathological and bacteriological reports were identical with those in the first case. Examination of immediate members of the family failed to reveal any others with similar lesions.[2a]

Treatment consisted of 46 gm. of streptomycin over a period of 23 days, after which granulations had disappeared, and cultures were negative for Klebsiella rhinoscleromatis. Follow-up of this patient through June, 1952, revealed no recurrence of the lesion, though there has been some tendency for crusting to take place.

SUMMARY

1. Two cases of scleroma from Panama have been presented, one in a native-born Panamanian.

2. Scleroma is not seen in Panama with the same frequency as in some of the Central American Republics.

3. Streptomycin has proved efficacious in the treatment of this disease in these cases.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Arthur J. Coombs, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

Jan. 7, 1952

Differential Diagnosis of Bronchial Asthma with Special Reference to the Upper Respiratory Tract. DR. LEON UNGER (by invitation).

The allergic person may have symptoms in any part of the respiratory tract. If the allergy affects the nose, we say that the patient has hay fever or perennial allergic rhinitis. In recent years allergic involvement of the sinuses has been included in one of these two categories. When the symptoms strike lower down, the patient has either bronchial asthma or allergic bronchitis.

The diagnosis of allergy is usually not difficult and is based on the following: (1) history of allergy in the family and/or other allergic conditions in the patient; (2) clinical tests whereby symptoms are relieved by removal of the causative factors and increased by reexposure; (3) positive skin tests which must be corroborated clinically; (4) symptomatic relief from such medicines as epinephrine or the antihistaminic drugs, and (5) the finding of eosinophiles in the nasal smear or sputum, as well as in the blood.

Tonight we are reporting on four patients who were referred because of symptoms suggesting bronchial asthma. Investigation, however, has shown that each one of these patients had some other condition rather than true allergic bronchial asthma. From these examples it is evident that the allergist must not jump to the conclusion that all the patients referred to him are allergic. It is also true that those who specialize in nose and throat work must always be on the lookout for patients whose symptoms result from allergy.

DISCUSSION

DR. PAUL H. HOLINGER: Dr. Unger has given us a most interesting discussion. It might be in order to ask if he would present this, or a similar paper, before the Allergy Society, also. It presents clearly the correlation between wheezing and intratracheal obstruction, and certainly from the standpoint of the otolaryngologist the association of the two must be kept in mind. Very frequently an obstruction accounts for the wheeze; an overlooked foreign body and bilateral bonchial obstruction are other conditions that fall into the category mentioned. I might mention one patient who had a persistent bilateral wheeze for approximately eight years, and finally x-ray examination demonstrated two large upholstery tacks, one in each bronchus.

The use of smears of sputum or bronchoscopically obtained secretions stained for eosinophiles frequently gives information as to whether the patient with bronchiectasis has a suppurative bronchial lesion alone or whether there is an allergic background. Dr. Hansel suggested that we use this diagnostic procedure, and we have found very definite clinical value to this test.

Dr. Unger mentioned the management of tracheal tumors. Very frequently endoscopic coagulation of the base of the tumor is of help, particularly if the tumor is not resectable. Otherwise, the newer forms of surgery may be helpful. Interesting work along this line is being done in other parts of the world, and recently in Honolulu we had the opportunity to see patients of Dr. Paul Gebauer, who has done resections of a portion of the tracheal wall, closing the defect by wire-supported fascial grafts, and has resected stenoses with anastomosis of the distal bronchi and lung to the bronchus proximal to the stenosis, resecting the portion that had been causing wheezing for a long period.

I should like to thank Dr. Unger again for presenting this interesting paper to us.

DR. LEON UNGER: I always enjoy hearing Dr. Holinger; he "hits the nail on the head." I would have no objection to giving this paper before the Society of Allergy. I think allergists

recognize their own faults, and the pendulum is beginning to swing in the other direction. I also remember, in a discussion before the Chicago Pediatric Society, the scorn with which my talk was received. Two or three of the men, however, were interested, and it was not too long before the pediatricians were doing skin tests, some well and some poorly. It is better to do none at all than not to do them well.

It is amazing how many rare and unusual allergens there are. Within the last two weeks we have had one patient sensitive to the castor bean and one to flaxseed; we have had patients sensitive to pyrogen, which is found in some wave-set lotions and hair tonics and also in linseed oil; that is why many patients have attacks while painting and decorating are being done. In Denver asthma occurred in many children in an institution while it was being decorated, and some of it must have been due to linseed oil.

So far as foreign bodies are concerned, we always look for them and ask about them in the history, but we have had only a few such cases in our office. I was glad to hear Dr. Holinger emphasize the importance of examining bronchial, as well as nasal, smears. If you use them for making autogenous vaccines, the question is whether they are better than ordinary stock respiratory vaccines. I think the effects are usually nonspecific.

I was interested in his remarks about surgery. Dr. Jerome Head, who removed the tumor in the case I presented, has discussed this with me, and I have left it in his hands. It seems logical, rather than to have the girl have recurrences.

The Treatment of Deafness by Prosthesis. Dr. H. G. Kobrak.

In certain sound-conduction lesions there is destruction of parts of or the whole ossicular chain. Attempts have been made to replace the missing eardrum and the destroyed ossicles by materials inserted into the tympanic cavity. The prothesis represents a simple, but frequently rather effective, method of improving hearing, and in its use new information may be gained as to the function of hearing. Three main groups of mechanical devices are described.

It is preferable that a natural closure of an eardrum perforation be accomplished, but if this cannot be achieved, several materials are adaptable to be used as prosthesis. The prosthesis may be left in place for several weeks without causing undue irritation. Supervision by the otologist, however, is indicated.

Any patient with a bilateral hearing defect due to destruction of the sound-conducting system should be investigated as to the feasibility of a tympanal prosthesis. It is an effective therapy, although its mechanism is not clearly understood. The vibratory behavior of the tympanal prosthesis can be observed during acoustic stimulation. [Motion picture demonstration.] Clinical experience has shown that the prosthesis is ineffective if the middle ear cavity is completely dry, and several methods have been devised for retaining moisture within the ear. Oxycholesterol-petrolatum (aquaphor®) ointment has been used in a series of cases with good effect, cases in which the round window niche was accessible and easily plugged. In other cases, especially in cavities left after radical mastoidectomies, the "waterbag" prosthesis was used, that is, a drop of water wrapped in latex membrane. Hearing gains up to 35 db. have been obtained. The prosthesis is invisible, of amazing simplicity, and negligible in cost, and, provided proper material is used, can be left in the middle ear for weeks or months.

DISCUSSION

Dr. Marvin Tamari: For the record only, I should like to say that about 60 years ago Politzer suggested an artificial drum to increase hearing. He also devised an instrument which could be handled by the patient himself. We started to experiment with the audiometer, and I found, in 1922, that by introduction of an artificial drum not only the hearing conduction but also the bone conduction could be improved by about 10 to 12 db.

Dr. H. G. Kobrak: We have frequently heard that other physicians have similarly good results with a prosthesis in the middle ear, and there are reports in the literature which are promising. Nevertheless, there are quite a few patients who have lived in the Chicago area all their lives, who have suffered from chronic middle ear infection all their lives, and who still have not been fitted with a prosthesis. I should like to express the hope that the profession will avail itself of this simple device for improving the hearing of patients.

A Cortical Test of Auditory Function in Experimentally Deafened Cats. Dr. Joseph E. Hind Jr. and Dr. Harold F. Schuknecht.

In an attempt to develop a test of function of the peripheral auditory system of the cat which would correlate well with the results of behavioral tests on the same animal, we have applied the method of evoked strychnine potentials from the auditory cortex, as first described by Tunturi (Tunturi, A. R.: Physiological Determination of Arrangement of Afferent Connections to Middle Ectosylvian Auditory Area in Dog, *Am. J. Physiol.* **162**:489, 1950). In this method, after the auditory cortex has been treated with strychnine sulfate solution, spike-like waves of electrical potential can be recorded from the cortex in response to the relatively abrupt onset of pure tones, and a determination can be made of the sound-pressure level required for threshold at all frequencies of interest. When the average threshold values obtained for a group of normal animals are compared with those for an animal with experimentally produced hearing loss, a curve may be obtained showing the increase in threshold sound pressure in decibels as a function of frequency. The experimental results presented compare the loss of function (increase in threshold sound pressure relative to the average normal value) for behavioral and cortical tests on seven animals. Five of the animals presented a high-frequency hearing loss due to lesions of the basal turn of the cochlea. The other two had partial section of the eighth nerve. The latter two cases are of hearing losses relatively severer for the low frequencies as a result of this partial section. They are of interest in that essentially normal aural microphonic potentials were recorded from the round window in both cats, while the behavioral and cortical results indicated a substantial loss in sensitivity for the frequencies below 8000 cps. A brief indication of how the functional test results correlate with histologic findings is also presented. The behavioral test will remain, by definition, the only true test of hearing. We find that the cortical and behavioral results agree well. The great savings in research time offered by the cortical method cause it to be a valuable tool in the experimental analysis of the peripheral auditory system.

Arthur J. Coombs, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

Feb. 4, 1952

The Treatment of Oroantral Fistulae. Dr. Linden J. Wallner.

For the purposes of management alveolar fistulae may be divided into two types: the acute, of recent origin, and the chronic of long duration.

The fundamentals of treatment in the acute type include adequate antimicrobial therapy, preferably penicillin, intranasal drainage of the associated sinusitis, and avoidance of manipulation of the fistula. With this regimen most acute oroantral openings will close spontaneously.

In chronic alveolar fistulae the pathologic conditions consist of an epithelized tract into the antrum, a chronic infection of the maxillary sinus, usually with anaerobic organisms, and some localized osteitis about the margins of the bony opening.

The requisites for successful closure of a chronic fistula are relief of the sinus suppuration by antrotomy combined with some type of plastic operation. This operation is best performed in two stages. The sinus surgery is carried out first, and when the antrum is free of infection, the fistula is closed by a plastic procedure.

The commonest plastic operation is the raising and fashioning of a flap from the hard palate and the rotating of it over the bony opening. Berger, in 1939, described a method of forming the flap from the lateral aspect of the alveolus. An incision is made over the fistula, and the edges are elevated to expose the size and location of the bony opening. The epithelial lining of the fistula is removed, with any softened bone. Formation of the flap is accomplished by making two divergent incisions at right angles to the first one and extending upward to the buccoalveolar fold. The flap is turned outward, and the periosteum is incised at the base, permitting free mobilization by utilizing the loose buccoalveolar fold. The flap is then secured over the bony opening without tension by means of mattress sutures, and the remaining edges are closed by single sutures. Details of the operation were illustrated by a motion picture.

Wallner has performed the Berger operation on 10 patients, with but one failure. The one unsuccessful result was in a patient who had the antrotomy and plastic operation carried out at the same sitting.

The Berger operation is readily performed. It seems especially adapted for fistulae located on the lateral aspect of the alveolus or those situated between two remaining teeth. Wider employment of this method is advocated.

The Nasal Mucosa of Allergic Patients Before and After Treatment with Corticotropin. DR. B. Z. RAPPAPORT, DR. M. SAMTER, H. R. CATCHPOLE, and DR. S. SCHILLER, with Technical Assistance of C. BADER (all by invitation).

The present study is an extension of a pilot experiment begun in 1950 on the effect of corticotropin (ACTH) in ragweed-sensitive patients during the hay-fever season. In the preliminary work (*J. Allergy* **22**:304-313 [July] 1951) nasal mucosa obtained by biopsy and stained by routine methods failed to show histologic differences in the tissues before and after corticotropin therapy. In the present study there was available the mucosa of 46 patients, 30 from the 1950 series and 16 from a new group seen during the August, 1951, season. Of these there were 32 patients whose tissues were obtained before and after corticotropin therapy and 16 who received no treatment and from whom biopsies were obtained for control study. All tissues were fixed by freezing in liquid air and drying *in vacuo*. Paired tissues were stained simultaneously by the periodic acid-leucofuchsin method which is highly specific for substances containing carbohydrates.

The mucosa obtained at the end of four days of treatment with corticotropin, when compared with that removed before treatment, showed consistently the following changes: (1) a general increase in stainability of the glycoprotein ground substance; (2) an increase in the cement substance between the epithelial cells of the mucosa; (3) an increase in the staining density and in the width of the submucous and of the perivascular "basement membrane"; (4) an increase in the number, the thickness, and the stainability of the fibers in the ground substance; (5) an increase in the number and size of red-staining granules in the cytoplasm of stellate, oval, and round cells present in the submucosa. These cells are believed to be undifferentiated fibroblasts (mesenchymal cells) and the granules to be glycoprotein in composition. It would appear from these results that during the allergic reaction the glycoproteins of the ground substance and the basement membranes are broken down (depolymerized) to smaller units with a proportionate loss in stainability. The effect of corticotropin is to arrest the process of depolymerization. The glycoproteins are restored toward the normal state with a resulting increase in stainability.

DISCUSSION

DR. O. E. VAN ALYEA: I have been much interested in this presentation. I know that corticotropin has been used during the active stages of hay fever with reported success, at the Mayo Clinic, at least. I wonder if Dr. Rappaport has formed any conclusions which will help us in a practical manner in treatment of nasal allergy of the seasonal or perennial type by the use of corticotropin or cortisone. If so, would it be justifiable in routine therapy, despite the possible ill effects that have been reported from the routine use of these hormones?

DR. JOHN J. BALLENGER: Dr. Henry L. Williams has noted that there is an increased capillary permeability in allergic states. He explained this condition by suggesting that there was a spasm perhaps of the arterioles with subsequent anoxia and dilatation of the distal capillaries and venules. The anoxia is presumed to be instrumental in producing the increased capillary permeability and resultant edema. I wonder if Dr. Rappaport has any information to corroborate or repudiate this.

DR. WALTER THEOBALD: A year ago someone in the East reported treating certain types of rhinitis by means of a cortisone nasal spray. I should like to ask Dr. Rappaport whether he has used corticotropin or cortisone in the form of a nasal spray instead of internal medication for these cases of allergic rhinitis.

DR. BEN Z. RAPPAPORT: Regarding the use of corticotropin and cortisone, it would seem to be an optimal means of treating an allergic reaction of short duration. Unfortunately, most allergic conditions are not of short duration; most patients are sensitive to many allergens. I have not emphasized clinical therapy. What impressed me about these people was their

great satisfaction, despite the small degree of improvement. I do not mean that they were not improved, but their happiness was out of proportion to the degree of improvement. There were patients with evident asthma, not severe but moderate, who said they felt wonderful.

A second point is that we are dealing with something we know too little about. If one is dealing with severe asthma that fails to respond to ordinary therapy, one should not hesitate to use corticotropin. One should, however, hesitate to use corticotropin for mild or moderate asthma. The temporary effect of the drug was apparent in this as well as in previous studies. Corticotropin was discontinued at the end of four days, and there was a rapid recurrence of symptoms within 48 hours. So far as its local use in the nose is concerned, we have had no experience with it. I have talked with those who have, and the reported results have been disappointing. I would not expect enough absorption of the material in view of the irritation and increased nasal secretion that results.

As for the reason for permeability of the blood vessels, the structural changes observed in the finer capillaries are amazing. The changes occurring in the many capillaries of the submucosa explain the rapidity of the edema. It should be noted that these changes are not limited to allergic conditions; similar changes occur in all types of inflammation. Regarding the question whether anoxia might explain the changes, one finds no evidence of anoxia. There is no vascular stasis. The rapidity of the changes (depolymerization) suggests an enzyme-like reaction. The return to normal following corticotropin therapy suggests an arrest in the breakdown process, permitting normal synthesis of glycoproteins (polymerization) to be reestablished.

Surgery in Intra-Oral Cancer. Dr. Maurice F. Snitman.

Prior to the advent of irradiation, cancer of the oral cavity was treated surgically and with comparatively poor results. The hope of increased curability following radiation therapy has not been realized. Consequently, surgery again is becoming the treatment of choice. Impetus to this modality has been provided by improvements in surgical technique and anesthesia, antibiotics, and a greater understanding of the importance of fluid and electrolytic balance. Additional measures, such as parenteral and catheter feedings and tracheotomy, have contributed greatly to this cause.

The histories of five patients are presented; these illustrate the variations in surgical procedure. In all cases the principle of the monobloc is adhered to. Five-year-cure rates are not as yet available. The improved immediate result, however, is demonstrated by absence of the almost constant excruciating pain that follows, in many instances, irradiation in one form or another. Surgical en bloc removal of the primary lesion, together with the regional lymph nodes and intervening lymph-bearing tissue, follows the fundamental principles laid down by Halsted 50 years ago when he described radical mastectomy.

DISCUSSION

Dr. Robert Lewy: It was my privilege to scrub in a number of the cases demonstrated tonight. I certainly regard this as progressive surgery. It is a step in the right direction that our newer techniques make this possible. It is a good thing that a few people are doing this type of work.

I should like to ask Dr. Snitman whether there are certain cases in which there is not a specific risk of metastases occurring to the contralateral side, and, if there are such cases, were they included in this series? If such cases exist, is this amount of surgery enough, or should not a suprahyoid dissection on the opposite side be required, or perhaps even more extensive surgery on the opposite side?

Dr. Hans Von Leden: I was very happy to hear Dr. Snitman's paper, because I agree so fully with everything he has said about this so-called monobloc or "commando operation," as it is called. There is one additional point on which I should like to comment. I have made it a point, in all cases in which suprahyoid nodes are positive on biopsy, to perform a radical neck dissection. If these nodes are involved, I feel that there is likelihood of involvement of other nodes as well.

Dr. M. F. Snitman: These patients are not being presented as cured. The two that had seed implantation of the primary lesion should not necessarily be examples for treatment along

these lines. Eradication of the primary lesion of the tongue by x-ray and radium therapy is generally unsatisfactory. The patient is usually left with an irradiation fibrosis, plus the residual fibrosis. The five patients operated upon are presented in the light of surgery of a similar nature in other parts of the body. It is scientifically correct, and the feeling of the men who have performed this procedure during the past few years is that there is a decided improvement over methods previously employed. So, we present these patients as immediate results.

We do not perform prophylactic neck dissections on such patients, since tongue and floor of mouth cancer in not a small number of cases develops crossed metastases.

There are only two kinds of neck dissection, so far as nodes are concerned: One is the suprahyoid, and that is always prophylactic in conjunction with surgical excision of oral cancer; the other, of course, is the total radical. When during a suprahyoid neck dissection positive metastatic nodes are encountered, a total radical neck dissection is the rule.

Stenosing Peptic Esophagitis. Dr. Stanton A. Friedberg and Dr. Frederic K. Clayton.

Esophagitis of mild degree is fairly common but may occasionally progress to the point where ulceration or low esophageal stenosis develops. Wilson (*Laryngoscope* **61**:423-447 [May] 1951) recently reemphasized this sequence in an excellent contribution. The subject has been previously analyzed by Chevalier Jackson, Allison, Benedict and Sweet, Olsen and Harrington, Vinson, Wangensteen, and others.

Most benign strictures which occur at the lower end of the esophagus are probably secondary to nonspecific esophagitis. It has been demonstrated both experimentally and clinically that esophagitis may result from the corrosive action of acid gastric secretion upon the esophageal mucosa.

Reflux of gastric secretion into the esophagus takes place when there is relaxation or incompetence of the cardiac sphincter, as in diaphragmatic hernias of the sliding type or when partial thoracic stomach is associated with a shortened esophagus having a congenital, spasmodic, or acquired basis. Peptic ulcer diathesis, chronic alcoholism, prolonged vomiting, moribund states, indwelling gastric tubes, or certain infectious diseases may also predispose to development of esophagitis.

The pathology and symptomatology of low esophageal stenosis resulting from peptic esophagitis are reviewed. Considerable responsibility rests with the endoscopist, not only in establishing the correct diagnosis but in carrying out the dilatations which control symptoms in the majority of the patients. Surgery of the esophagus and stomach offers a solution for intractable cases in which failure of dilatation, recurrent hemorrhage, persistent pain, or esophageal perforation complicates the clinical picture.

Case histories of six patients with stenosing peptic esophagitis illustrate the occurrence of peptic ulcer diathesis in all six, the high incidence of chronic alcoholism (five patients), and hiatal hernia of the congenital type in one patient and of the acquired form in four others. An interesting side-light was the spontaneous perforation of gastric or duodenal ulcers in two patients during symptom-free intervals between dilatations for their esophageal strictures.

DISCUSSION

Dr. Paul H. Holinger: Dr. Friedberg has added another important contribution to his previous work on the esophagus and, together with Dr. Clayton, has presented an interesting group of cases. I should like to ask a question with regard to two patients: In the first patient the stricture was so extremely high that I wonder why they classify this as a peptic ulcer of the esophagus at 27 cm. from the upper teeth and whether or not there was some other information aside from that given which caused them to consider this as a peptic ulcer. I was a little confused about the last patient with regard to the nature of his lesion: Why was the plastic operation on the esophagus followed by further esophagoscopic dilatations? If further dilatations were necessary, had a stricture again developed at the point of the anastomosis, or did the reflux of secretion from the stomach cause another peptic ulcer to develop with further stenosis? If a postmortem examination was done, what did it show with regard to the lesion in the esophagus?

DR. STANTON A. FRIEDBERG: Actually, some of these patients were admitted with a tentative diagnosis of carcinoma of the esophagus and did offer diagnostic problems. When presented in sequence like this, the clinical picture of esophagitis and benign stenosis becomes fairly clear, but for the sake of the record it should be emphasized that the correct diagnosis was not immediately evident in the earliest cases of the series.

In reply to Dr. Holinger's first question regarding the patient with the congenital short esophagus it should be pointed out that a good deal of time was spent in trying to determine the etiology of his stricture. He had no severe febrile illness and no history of caustic ingestion; in fact, we practically accused him of not telling the truth about his illness. We finally concluded from biopsy specimens obtained above and below the stricture that he did have a short esophagus, peptic esophagitis, and a benign stricture. The second patient was an even greater problem. He had been operated upon so many times in the past that the surgeons were reluctant to attempt any further surgery. It was unfortunate that we were unable to obtain a postmortem examination.

Wangensteen is of the opinion that esophagitis is so commonly associated with peptic ulcer as to be considered part of the ulcer syndrome, and he feels that esophagitis may be the mildest form of the ulcer diathesis. It is true that dilatation of these strictures poses a dilemma in that, after relief of the stenosis, gastric secretion flows more readily into the esophagus. However, esophagitis is less troublesome than a persistent stricture, and the final answer will doubtless depend upon actual solution of the whole ulcer problem.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF APRIL 1, 1953

Audicon Models 400, 415, 530, & 615

Mfr., National Earphone Co., Inc.
20 Shipman St.
Newark 2, N. J.

Audiotone Model 11

Mfr., Audio Company of America
5305 N. Sixth St.
Phoenix, Ariz.

Audivox Model Super 67

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L & M

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"

Beltone Symphonette Model

Mfr., Beltone Hearing Aid Co.
1450 W. 19th St.
Chicago 8

Cleartone Models 500 & 700

Cleartone Regency Model

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3

Dahlberg Model D-4

Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Dysonic Model 1

Mfr., Dynamic Hearing Aids, Inc.
149 Church St.
New York 7

Electro-Ear Model C

Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Models V-35 & V-60

Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97

Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corporation
708 W. 40th St.
Minneapolis 8

Maico Model J

Maico Quiet Ear Models G & H

Maico Top Secret Model L

Maico UE Atomeer

Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal & Magnetic) Aurophone Model 200

Mfr., Mears Radio Hearing Device Corporation
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10, T-612, & 45
Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8
Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Model E-4

Otarion Models F-1, F-2, & F-3

Otarion Models G-2 & G-3

Otarion Model H-1
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC, & YC-7) (Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear All-Magnetic Model 55

Radioear Permo-Magnetic (Multipower)

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)

Radioear Permo-Magnetic (Uniphone)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mount Lebanon
Pittsburgh 16
Distr., Radioear Corporation

Rochester Models R-1 & R-2
Mfr., Rochester Acoustical Laboratories, Inc.
117 Fourth St., S. W.
Rochester, Minn.

Silvertone Models J-92 & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck and Co.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, & 966
Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 97, 99, 200, 300-B, 400, 500, 952, 953, & 1700

Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model

Tonemaster Model Royal

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2

Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 & 66

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"

Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)

Mfr., A. N. Brooks Company
684 S. Bonnie Brae St.
Los Angeles 5

Aurex Semi-Portable

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid

Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50

Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

CEREBROSPINAL RHINORRHEA FROM CRANIOMASTOID FISTULA. R. H. AMES, W. D. FARMER, and W. H. MILLS, A. M. A. Arch. Otolaryng. **53**:451, 1951.

Leakage of cerebrospinal fluid from the nose is by no means a rare sequel of head trauma. Such a finding is generally regarded as pathognomonic of a communication in the region of the paranasal sinuses or the cribriform plate. It has been shown, however, that in rare instances rhinorrhea may result from a communication between the subarachnoid space and the mastoid or middle ear, the fluid passing through the Eustachian tube and into the nasopharynx.

The authors report such a case of cerebrospinal fluid rhinorrhea resulting from a traumatic craniomastoid fistula in a child of 6 years. Preoperative identification of the site of leakage by catheterization of the Eustachian tube enabled the correct surgical approach to be made and prevented a misdirected exploration of the anterior fossa.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

STUDIES IN SHORT DURATION AUDITORY FATIGUE: V. AN INVESTIGATION OF THE SPREAD OF FATIGUE WITHIN NARROW FREQUENCY LIMITS. ANITA I. RAWNSLEY and J. DONALD HARRIS, Bureau of Medicine and Surgery, Navy Department, Project NM 003 041.34.04 XI (16) (May 16) 1952.

Auditory fatigue to 1000 cps tones of 30, 50, 70, and 90 db. sensation level was determined in five observers, and its spread to closely adjacent frequencies was examined. The method of residual fatigue was used to avoid the intrusion of beats in simultaneous masking. When a tone was sounded in one ear and immediately a second tone was sounded, there was a decrease in sensitivity to the second tone. The largest decrement was found for test tones from 990 through 1010 cps, with a broad spread at higher sensation levels.

PALMER, Wichita, Kan.

EXPERIMENTS ON FLUCTUATION OF AUDITORY ACUITY. J. DONALD HARRIS and CECIL K. MYERS, Bureau of Medicine and Surgery, Navy Department, Project NM 003 041.21.06 XI (13) (June 22) 1952.

Auditory thresholds were determined on 3 men in 1-db. steps as a function of time: moment-to-moment, hour-to-hour, day-to-day, and week-to-week. The typical moment-to-moment fluctuation was of the order of slightly less than 1 db. This finding is not in accord with the views of those who feel the instability of the audiogram is in the order of ± 5 decibels. Relatively greater variability of high tone acuity as against low tone acuity was determined to arise not from inexact head phone placement but probably from an instability of the initial section of the basilar membrane.

PALMER, Wichita, Kan.

MASKING AND FATIGUE EFFECT OF WHITE NOISE IN CONNECTION WITH SPEECH TESTS. CARLO SAMBATARO, and GIULIO PESTALOZZA, Laryngoscope **62**:1197 (Nov.) 1952.

The effects of "white" noise on hearing thresholds are shown, with use of a free field and logatomes. There was a much lower threshold shift for low intensities of the white noise taken with higher intensities, where the threshold shift corresponded to the intensity of the noise itself. This fact is confirmed by a person's being able to hear on the long-distance telephone in the presence of a low background noise.

The fatigue effect of white noise was also studied. Surprisingly, it was found that the threshold was the same or even slightly lower after the ear was exposed to the noise (60 to 80 db.) for 15 minutes. Hypotheses are given to try to explain this phenomenon.

HITSCHLER, Philadelphia.

THE SEVENTH CRANIAL NERVE IN ACOUSTIC NEUROMA. C. AMBROSETTO, Riv. oto-neuro-oftal. **26**:549 (Nov.-Dec.) 1951.

Involvement of the seventh cranial nerve is exceptional in cases of acoustic neuroma. Mild and partial damage is encountered more frequently. Ambrosetto reports his observations on the impairment of the seventh nerve in 17 verified cases of acoustic neuroma. In two cases with relatively large tumors there was no evidence of involvement of the seventh nerve. In one case with a long history and a large tumor, with increased intracranial pressure, involvement of the seventh nerve was limited to mild weakness of the orbicularis muscle of a few months' duration. In seven cases there was moderate evidence of impairment of seventh-nerve function. In four of these seven cases all three divisions were involved; in two cases only the third, and in one case only the first. In two instances with mild weakness of the facial nerve there were clonic twitches of the face, limited to the chin in one and involving the whole face in the other. In one case there was a mild increase of tone in the facial muscles on the side of the tumor; this increase was evident at rest and was associated with weakness in the third division. In two cases only mimetic facial weakness was noted on the side of the neoplasm. In one case there was a marked paresis of all three divisions of the seventh nerve, with involvement of the other cranial nerves of the posterior fossa on the same side. Taste was investigated in nine cases; it was impaired in the anterior two-thirds of the tongue in four and on the whole of the side of the tongue ipsilateral to the tumor in one. There was no clear relation between degree of involvement of the fifth and that of the seventh cranial nerve. The relative sparing of the seventh nerve is due to the fact that in the region of the angle it can be readily displaced by slowly growing tumor tissue because of its looseness and mobility.

N. SAVITSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

THE QUESTION OF OCCULT MASTOIDITIS IN INFANTS AND POSSIBLE CURE WITH PENICILLIN. KURT NITSCH, Monatsschr. Kinderh. **97**:449-452 (Jan.) 1950.

In infants severely ill with nutritional disturbances, generalized toxicity, vomiting, fever, foul stools, and the findings of mild thickening, injection, or retraction of the tympanic membranes, mastoidectomy should be performed if other treatment, including three to four days of penicillin therapy, prove unsuccessful. These infants usually come from poor unhygienic homes but may be occasionally quite well nourished. Of 42 patients with what the authors called otitis with occult retrotympanic suppuration, 35 underwent surgery and 21 were cured.

HARTENSTEIN, Madison, Wis. [A. M. A. AM. J. DIS. CHILD.].

Larynx

RECURRENT LEFT LARYNGEAL NERVE PARALYSIS IN ARTERIOSCLEROTIC HEART DISEASE. S. ZELMAN and G. W. NIE, J. A. M. A. **149**:1291 (Aug. 2) 1951.

Hoarseness due to paralysis of the left recurrent laryngeal nerve is a rare occurrence in patients with congestive heart failure due to any cause, but it is most commonly seen with severe mitral stenosis, and less commonly with congenital heart disease, when marked dynamic dilatation of the left pulmonary artery compresses the nerve against the aortic arch, its adjacent lymph nodes, and the ligamentum arteriosum. It is extremely uncommon in patients with arteriosclerotic heart disease, since the primary cardiac strain in such patients is nearly always on the left ventricle. Only when the arteriosclerotic left ventricle fails does increased pressure occur in the left auricle and the pulmonary vessels.

Zelman and Nice describe such a case in which left ventricle failure had been continuous, though varying in degree, for nearly four years. The patient was admitted on two occasions during that time with evidence of failure and, in addition, complete paralysis of the left vocal

cord. While digitalization and treatment with mercurial diuretics did not entirely relieve his left heart failure, the hoarseness and paralysis of the vocal cords cleared repeatedly.

An additional factor in the compression of the recurrent laryngeal nerve in this case may have been the unusually low position of the aortic arch in relation to the left pulmonary artery.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

LARYNGECTOMIES IN REVIEW: 20 YEARS' EXPERIENCE. FRANCIS E. LEJEUNE and MERCER LYNCH, Selected Writings of the Ochsner Clinic **11**:1 (June 30) 1952.

LeJeune and Lynch have followed up a series of 537 patients in their private practice during a period of 20 years. Of this group, 172 patients (32%) had such extensive lesions that they were considered inoperable. This finding led the authors to emphasize once more the necessity of educating the medical profession and the public regarding the significance of the early symptoms of laryngeal cancer. These patients were advised to take a course of irradiation, and those who accepted this treatment prolonged their lives.

In 194 patients the lesions were small enough for intralaryngeal extirpation or laryngofissure, whereas in the remaining 171 (31.7%) the lesions were so far advanced that laryngectomy was necessary.

The authors discuss the incidence according to age, sex and color and the symptomatology, diagnosis, indications, technique, and results.

Laryngectomy should be performed only on patients who are in good or fair physical condition; hence a thorough physical examination is a preoperative requisite. The importance of eliminating cardiorenal, diabetic, and metastatic complications cannot be too strongly emphasized.

It was found that laryngectomized patients readjust nicely to their new mode of life, and the majority usually learn the fundamentals of an esophageal voice; the development of such speech is limited only by their initiative.

GORDON, Philadelphia.

RECURRENT LARYNGEAL PARALYSIS IN CARDIAC DISORDERS: ORTNER'S SYNDROME. A. QUADRELLI, Prensa méd. argent. **39**:293 (Feb. 15) 1952.

Ortner reported laryngeal paralysis in association with mitral stenosis for the first time in 1897. This paralysis was considered due to pressure on the recurrent laryngeal nerve by the dilated left auricle and the aortic arch. Alexander, in 1904, suggested that laryngoplegia is due to compression of the recurrent laryngeal nerve by a dilated left pulmonary artery. Additional pressure against the nerve was considered possible by the pushing upward of the dilated auricle against the pulmonary artery. The author reports two cases of heart disease with recurrent laryngeal paralysis. In both cases there was evidence of heart failure with passive congestion of the lungs. One patient was a woman aged 45 with mitral stenosis; the other, a woman aged 54 with hypertension and heart failure; both had laryngeal paralysis. In the second the left auricle was not enlarged. The author considers that the recurrent laryngeal nerve involvement in both cases was due to pressure from a dilated left pulmonary artery. The nerve is compressed in the triangle formed by the arch of the aorta, superiorly, the left pulmonary artery, below, and the ligamentum arteriosum, medially. The dilated auricle in mitral stenosis may also play an accessory role by pushing the dilated pulmonary artery upward.

N. SAVITSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

Miscellaneous

TREATMENT OF PNEUMOCOCCAL MENINGITIS WITH LARGE DOSES OF PENICILLIN. P. A. BUNN and G. PEABODY, A. M. A. Arch. Int. Med. **89**:736 (May) 1952.

In 1941 Dowling and his group reported their observations on a series of 21 patients with pneumococcic meningitis who were treated with large doses of parenterally administered penicillin. Their observations indicated that a regimen of 1,000,000 units given by the intramuscular route every two hours resulted in an appreciable drop in mortality. The incidence of complicating sequelae and the total length of illness were also reduced.

Bunn and Peabody present a series of 20 patients with bacteriologically proved pneumococcic meningitis who were similarly treated with large doses of parenterally injected penicillin. They used and recommended the administration of a dose of 1,000,000 units every two hours for at least one week, or until clinical remission seems secure. They feel that the intrathecal use of penicillin is not desirable, for the reason cited by Dowling, viz., poor penetration to all areas of infection and the irritative qualities of the drug. They found that adjuvant therapy with a sulfonamide or aureomycin apparently adds nothing to the efficacy of the regimen, but point out that it is not contraindicated.

Of the 20 patients in this series, 15 recovered and 5 died, 3 within 24 hours of hospitalization. None of these three received more than 12 doses of penicillin. The other two patients, aged 82 and 83, died after receiving therapy for 12 and 15 days, respectively. In both patients evidences of meningeal infection had subsided, and death was due to other causes.

Fifteen patients survived and were free from all evidence of meningitis. Residua referable to the central nervous system occurred in only two patients. The seventh-nerve paralysis in one was not of major importance and did not preclude a complete return to active life. The vestibular and auditory defects in the second patient gradually cleared, and after six months there was no significant loss of either function.

Twelve surviving patients in this series received therapy for 6 to 19 days. Treatment was extended 24 to 40 days for three other patients because of the persistence of chronic middle ear infection and abscess of the eye. During the period of acute meningitis, spinal fluid concentrations of 0.05 to 0.5 unit per cubic centimeter were obtained with 1,000,000 units of penicillin given intramuscularly. Blood concentrations reached peak levels of 5 to 20 units with this dose.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

EXTRADURAL CEREBELLAR HEMATOMA. A. J. BELLER and E. PEYSER, J. Neurosurg. **9**:291 (May) 1952.

The authors present three cases of extradural cerebellar hematoma; in one the operation was successful whereas in the other two the outcome was fatal. The literature on this subject is reviewed, and the clinical features and main findings are discussed.

Beller and Peyser point out that epidural bleeding into the posterior fossa of the base of the skull should be suspected in cases of trauma to the back of the head when signs of increased intracranial pressure and abnormal neurological findings, either cerebellar or noncerebellar, appear together after a lucid interval, which, however, need not invariably be present. The condition usually develops more slowly than epidural hematoma in the middle fossa, since the bleeding is always venous. External signs of injury, as well as plain roentgenograms, may be helpful in the correct diagnosis and exact localization of the lesion.

Rigidity of the neck and cerebellar signs usually indicate a lesion in the posterior fossa. Pyramidal tract signs, whether homolateral or contralateral, neither exclude nor prove this diagnosis. Occasionally a sign of third nerve involvement occurs, but should not lead to the exclusion of an infratentorial lesion. Although no definite explanation of this phenomenon can be given, that of pressure on the nerve from below is most likely.

The only therapeutic measure which may offer success is surgical removal of the clot and sealing off of the origin of bleeding. The authors advocate infratentorial exploration in any case in which the history and clinical findings arouse suspicion.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

THE DIFFERENTIATION OF INTERIORIZED AND EXTERIORIZED SECONDARY STUTTERING. ERNEST DOUGLASS and BRUCE QUARRINGTON, J. Speech & Hearing Disorders **17**:377-385 (Dec.) 1952.

Stutterers can be divided into two types: those who attempt to mask their stuttering, and those who attempt to avoid it by devices. The interiorized stutterer wants to avoid stuttering. The exteriorized stutterer wants to express himself. Interiorized stutterers appear to come from families that are upwardly oriented and employ child-rearing practices and methods of discipline particularly suited to children who are to maintain the social status of the family. Case histories show that the families of interiorized stutterers showed earlier and greater concern for speech behavior.

PALMER, Wichita, Kan.

VOCAL RESPONSES DURING CONTROLLED AURAL STIMULATION. CHESTER J. ATKINSON, J. Speech & Hearing Disorders **17**:419-426 (Dec.) 1952.

Twenty-four males read 90 monosyllables during successive exposures to tones of varying loudness and frequency given three seconds before the speaker read each syllable The sound-pressure level of vocal responses tended to increase as the loudness level was increased. Frequency of the stimulus did not affect sound-pressure level. Loudness level of stimulus raised the response frequency. Frequency of stimulus tone had no effect on fundamental frequency in voice. Speakers change their voices in response to differences in the acoustic environment relative to loudness of the stimulus tones in that environment.

PALMER, Wichita, Kan.

CORROSIVE STRICTURES OF THE ESOPHAGUS AND THEIR TREATMENT. MERCER G. LYNCH, Selected Wrintings of the Ochsner Clinic **8**:24 (Dec. 31) 1949.

Lynch states that the numerous methods of treating corrosive strictures of the esophagus have met with varying degrees of success, but, despite legislation requiring labeling of all caustic substances and despite the fact that lye as a household agent is much less popular than formerly, strictures of the esophagus produced by lye still occur too often.

He reports three cases with roentgenograms. Details are given in the first 24 hours of the acute stage, when the pediatrician or internist first sees the patient, after which the otolaryngologist must plan an exact management of the case.

Retrograde esophagoscopy with through-and-through passage of the string is not an easy task and often is a risky procedure. Accomplishment of this step, however, marks a distinct milestone in the progress of the patient. Passage of the bougie from above for the first time is accomplished by means of the fluoroscope, which is safe. Progress is slow. In longitudinal stricture dilatations just about "hold their own." Deep burns of the esophagus cause dense scar tissue, and this requires repeated dilatations indefinitely. Where the burn is so extensive that even a filiform catheter cannot be passed, gastroesophagoscopy is the only method that will enable the patient to return to a normal mode of life.

GORDON, Philadelphia.

THE PROBLEM OF THE DIZZY PATIENT. MILES L. LEWIS JR., Selected Writings of the Ochsner Clinic **10**:30 (Dec. 31) 1951.

Lewis discusses the manifold factors of this important problem. He reviews the peripheral origin affecting the vestibular end-organs and the eighth cranial nerve, as well as the likely conditions of the external auditory canal, the Eustachian tube, allergy, inflammatory or neoplastic states, fracture of the temporal bone, the blood supply, Ménière's disease, and endolymphatic hydrops.

Vertigo of central origin is not as severe as that of peripheral origin, and it may be due largely to a diminished blood supply to the vestibular nuclei and tracts. Numerous other causes may be responsible.

It is of the utmost importance that the otologist should determine what the patient means when he says he is dizzy. Milder positional vertigo is usually of central origin.

The problem calls for an accurate history, a complete examination of the ear, nose, and throat, including evaluation of the labyrinthine and auditory functions, a thorough medical examination, and even neurological and ocular surveys.

GORDON, Philadelphia.

HEADACHE, SYNCOPE AND VERTIGO. L. S. MERIWETHER, W. S. CULPEPPER, and S. W. TUTHILL, Selected Writings of the Ochsner Clinic **11**:20 (June 30) 1952.

Headache, syncope, and vertigo are common symptoms of numerous pathologic states and conditions of disturbed physiology. This article discusses in moderate detail the headache due to localized space-restricting lesions, vascular conditions, intracranial infectious diseases, febrile infectious systemic diseases, lesions of teeth, eyes, ears, nose and neck, spinal puncture headache, post-traumatic headache, head pain from neuritides, and nervous tension. Under each heading, near the end of any given topic, one will find suggested therapy.

The symptoms of syncope and vertigo are discussed in the same manner. Each case requires a thorough work-up in order to obtain a true evaluation.

GORDON, Philadelphia.

VERTIGO AND CERVICAL ADENOPATHY. MAYOUX, CHOSSEGROS, and LAFON, Rev. oto-neuro-opht. **24**:65 (Feb.-March) 1952.

The authors report three cases in which a fairly clear relation existed between attacks of vertigo and cervical adenopathy. In the first case vertigo recurred each time there were swelling and flaring-up of a tuberculous cervical node. In the second case vertigo disappeared after a course of radium therapy to the cervical node, involvement of which appeared secondary to epithelium of the face. In the third case vertiginous attacks disappeared after excision of a calcified node near the carotid artery. The authors believe that the attacks of vertigo are a result of involvement by the enlarged node of autonomic fibers which go to the inner ear. During an attack of vertigo in the first case retinal arterial pressure was 35 mm. on the left and 50 mm. on the right. Eight days after the vertiginous attack the retinal arterial pressure was equal on the two sides. The change in retinal arterial pressure is added evidence that during the vertiginous seizure there was significant impairment of the intracranial vasomotor system. In the third case carotid sinus dysfunction as a possible cause of the vertigo is suggested. The authors did not find any other similar cases in the literature.

N. SAVITSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

News and Comment

ANNUAL SESSION OF THE AMERICAN MEDICAL ASSOCIATION
New York, June 1-5, 1953

The following portion of the program of the American Medical Association Scientific Session relating to laryngology, otology, and rhinology will be of interest to readers of this Journal.

SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

JADE ROOM, WALDORF-ASTORIA HOTEL

Tuesday, June 2—2 p. m.

Introduction of Scientific Exhibitors — Francis W. Davison, Danville, Pa.

Symposium on the Management of Chronic Sinus Disease

Management of Chronic Maxillary Disease — Lawrence R. Boies, Minneapolis

Management of Chronic Ethmoid and Sphenoid Sinusitis — Fred W. Dixon, Cleveland

Management of Frontal Sinus — Lewis Francis Morrison, San Francisco

The Frontal Sinuses in Relation to Trauma: Initial and Subsequent Cure
Walter P. Work, San Francisco

Motion Picture: Commando Operation for Carcinoma of Head and Neck
Hans Von Leden, Evanston, Ill.

Wednesday, June 3—2 p. m.

BUSINESS MEETING

ELECTION OF OFFICERS

Chairman's Address: Surgical Treatment of Persistent Thyroglossal Ducts and Cysts
Dean M. Lierle, Iowa City

Reaction to the Nasal Mucosae: The Relation of Life Stress to Chronic Rhinitis and "Sinus" Headache — Stewart Wolf, Oklahoma City

Discussion to be opened by Edmund P. Fowler Jr., and George E. Daniels, New York

Cancer of the Larynx: Laryngoplasty to Avoid Laryngectomy
Joel J. Pressman, Los Angeles

Discussion to be opened by De Graaf Woodman and John F. Daly, New York

Technique on Removal of Adenoid Tissue of the Nasopharynx in Children
Philip E. Meltzer, Boston

Discussion to be opened by Joseph L. Goldman, New York, and Ernest Reeves, Passaic, N. J.

Thursday, June 4—2 p. m.

Otitis Externa: The Facts of the Matter — J. W. McLaurin, Baton Rouge, La.

The Treatment of Traumatic Deformities of the Face
John Marquis Converse, New York

Discussion to be opened by V. H. Kazanjian, Boston, and Thomas William Stevenson, New York

The Fenestration Operation Analyzed for the Nonfenestrating Otologist

J. BROWN FARRIOR, Tampa, Fla.

Discussion to be opened by CLAIR M. KOS, Iowa City, and ARTHUR L. JUERS, Miami, Fla.

Otosclerosis in Identical Twins: Factors in Onset, Exacerbation and Stabilization

EDMUND PRINCE FOWLER SR., New York

Discussion to be opened by FRANZ ALTMANN and RAFAEL LORENTE DE NÓ, New York

SECTION ON ANESTHESIOLOGY

JADE ROOM, WALDORF-ASTORIA HOTEL

Tuesday, June 2—9 a. m.

Endotracheal Anesthesia in Children for Tonsillectomy and Adenoidectomy and for Other Surgical Procedures of Longer Duration

JOHN W. PENDER, Rochester, Minn.

Discussion to be opened by SAMUEL L. LIEBERMAN, Buffalo; A. H. KORNBLAU, Jamaica, N. Y., and ROBERT M. SMITH, Boston

Histological Changes in Nerve and Soft Tissues After Injection of Various Local Anesthetic Substances

WALTER H. MANNHEIMER, JOHN ADRIANI, and PHILIP PIZZOLATO, New Orleans

Discussion to be opened by DANIEL C. MOORE, Seattle, and ALBERT M. BETCHER, New York

Cerebral Edema and Its Relationship to Barbituric Acid Poisoning

LLOYD H. MOUSEL, Seattle

Discussion to be opened by RAYMOND F. COURTIN, Worcester, Mass.; RICHARD H. BARRETT, Hanover, N. H., and DONALD E. HALE, Cleveland

ANNOUNCEMENTS

Wanted—Head of Ear, Nose, and Throat Department at Christian Medical College, Vellore, South India.—The Vellore Christian Medical College, South India—eighty miles west of Madras City—is an international, union enterprise of the mission boards of Protestant Church denominations in Canada, Britain, Australia, and the United States. The medical school, which was originally established in 1916, is affiliated with the University of Madras, which confers the M.B.B.S. degree—Bachelor of Medicine, Bachelor of Surgery, equivalent to M.D. in the United States. The medical college hospital has 550 beds, a dozen of which are allotted to the department of otolaryngology. The ear, nose, and throat outpatient department has a daily attendance of 50 to 60 patients.

There are 175 students enrolled in the five medical classes. The program of teaching comprises 12 lectures to the fifth-year students, sections for clinical work, rounds, and clinico-pathological conferences. The total faculty consists of about 60 full-time teachers, of whom about a third are Westerners. On the ear, nose, and throat staff there are an American professor, now giving part time and desiring to withdraw; an associate who has the Diploma in Oto-Rhino-Laryngology of the University of Madras; an intern, and two residents.

There is a large volume of clinical and operative work, dealing with a great variety of conditions—some peculiar to Southern Asia, many the result of neglect and the lack of sulfonamides and antibiotics in the villages.

The applicant should be a member of a Protestant church, a Diplomate of the American Board of Otolaryngology, have three years of teaching experience (training of interns and residents in a university-affiliated hospital accepted), and be willing to serve for a period of at least three years, on a very modest salary.

Further details may be obtained from Miss Sarah Scudder Lyon, Executive Secretary, Vellore Christian Medical College Board, 156 Fifth Avenue, New York 10.

American Academy of Ophthalmology and Otolaryngology.—The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1, and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, Executive Secretary-Treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

Request for Reprints.—The following letter, reproduced in its entirety, was received from Dr. Walter D. Chase, 230 E. Broad St., Bethlehem, Pa.

To the Readers of A. M. A. ARCHIVES OF OTOLARYNGOLOGY:—Members of our profession behind the Iron Curtain find difficulty in learning of medical progress in the world beyond their boundary. When opportunity for widening their horizon occurs, I am sure all of us welcome such a chance to contribute to keeping them up to date.

Dr. George J. Figlear, dental surgeon, 124 E. 4th Street, Bethlehem, Pa., has been asked by a physician in Czechoslovakia to try to supply him with information on our operative technique in oto-rhino-laryngology. I believe he will be most informed if he can receive reprints on this work from all of us who have published articles on our surgical technique. Dr. Figlear will be happy to receive all such reprints and forward them to his deserving friend.

I hope for a wide response, as a gesture of goodwill to physicians behind the Iron Curtain.
Yours appreciatively,

WALTER D. CHASE

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont. Time: June 14-18, 1953.

Fifth International Congress of Oto-Rhino-Laryngo-Broncho-Esophagology

Place: Amsterdam. Netherlands. Time: June 8-13, 1953.
President: Prof. Dr. Eelco Huizinga.
General Secretary: W. H. Struben, Viottastraat 1, Amsterdam-Z
Scientific Secretaries: Dr. J. Bijtel and Dr. P. G. Gerlings.

International Bronchoesophagological Society

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Second International Congress of Bronchoesophagology.
Place: Albert Plage, Le Zoute, Belgium. Time: June 15-16, 1953.

International Congress of Audiology

Place: Leyden, Netherlands. Time: June 5-6, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland

International Course in Paedo-Audiology

Place: Groningen, Netherlands. Time: June 1-4, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico. Time: Feb. 28-March 4, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: Feb. 21-25, 1954.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James Milton Robb, 315 Lakeland Ave., Grosse Pointe 30, Mich.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 11-17, 1952.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Roosevelt Hotel, New Orleans. Time: April 21-25, 1953.

American Broncho-Esophagological Association

President: Dr. Fred W. Dixon, 1027 Rose Bldg., Cleveland.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Roosevelt Hotel, New Orleans. Time: April 28-29 (afternoons only).

American Laryngological Association

President: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 7.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Roosevelt Hotel, New Orleans. Time: April 26-27, 1953.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis E. LeJeune, 49 Audubon Blvd., New Orleans 18.
President-Elect: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Roosevelt Hotel, New Orleans. Time: April 28-30, 1953 (mornings only).

SECTIONS:

Eastern.—Chairman Dr. Irl H. Blaisdell, 1100 E. Genesee St., Syracuse 10, N. Y.
Place: Hotel Syracuse, Syracuse, N. Y. Time: Jan. 7, 1953.
Southern.—Chairman: Dr. W. W. Wilkerson, Jr., Curtiswood Lane, Nashville 4, Tenn.
Place: Andrew Jackson Hotel, Nashville, Tenn. Time: Jan. 12, 1953.
Middle.—Chairman: Dr. Henry L. Williams, 818 4th St., S. W., Rochester, Minn.
Place: The Drake, Chicago. Time: Jan. 19, 1953.
Western.—Chairman: Dr. Alden H. Miller, 4000 Woking Way, Los Angeles 27.
Place: The Elks Club, Los Angeles. Time: Jan. 24, 1953.

American Otological Society, Inc.

President: Dr. Albert C. Furstenberg, 201 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Roosevelt Hotel, New Orleans. Time: May 1-2, 1953.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Louis Joel Feit, 50 Park Ave., New York 16.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago. Time: Oct. 16, 1953.

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8.	Barbizon (For Women) 140 East 63rd St.	4.75- 5.50	8.00- 8.50
12.	Barbizon-Plaza, 101 West 58th St.	6.50- 8.50	11.00-14.50
47.	Barclay, 111 East 48th St.	Suites only	
62.	Beaux Arts, 310 E. 44th St.	6.50- 8.50	8.50-12.00
43.	Beekman Tower, First Ave. & 49th St.	6.00- 8.00	7.00-12.50
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45.	Bristol, 129 West 48th St.	....	7.00- 8.50
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65.	Claridge, Broadway & 44th St.	6.00- 8.00	8.00-11.00
70.	Commodore, Lexington Ave. & 42nd St.	6.50- 9.50	10.50-14.00
64.	Dixie, 250 West 43rd St.	5.00- 6.00	8.00- 9.50
11.	Essex House, 160 Central Park South	8.00-13.00	11.00-17.00
80.	Governor Clinton, Seventh Ave. & 31st St.	6.00- 7.50	8.50-12.00
83.	Gramercy Park, Lexington Ave. & 21st St.	5.00- 9.00	7.00-12.00
19.	Great Northern, 118 West 57th St.	6.00- 7.00	8.00-10.00
84.	Grosvenor, 35 Fifth Ave.	7.00- 9.00	10.00-12.50
18.	Henry Hudson, 353 West 57th St.	5.00- 7.00	8.00-12.00
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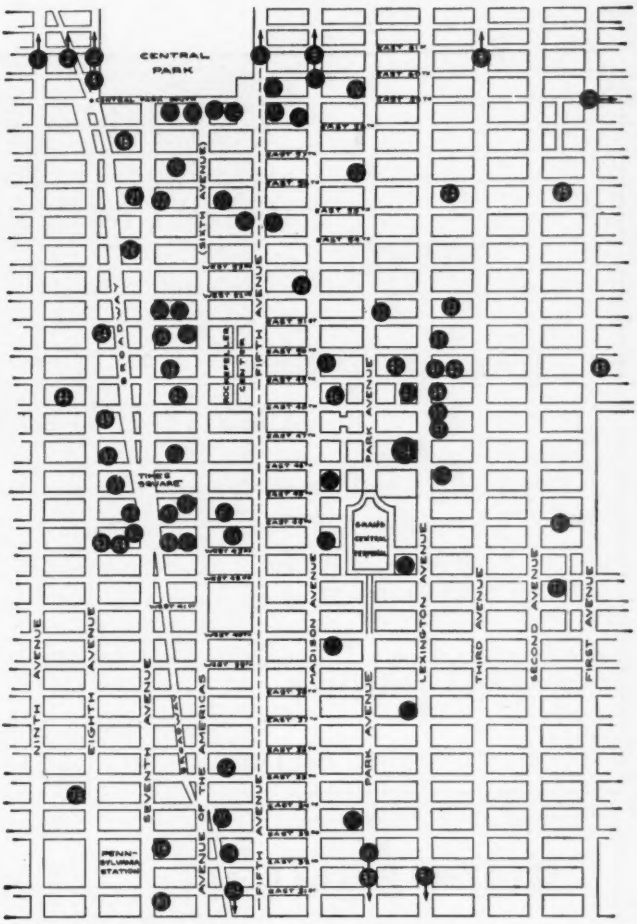

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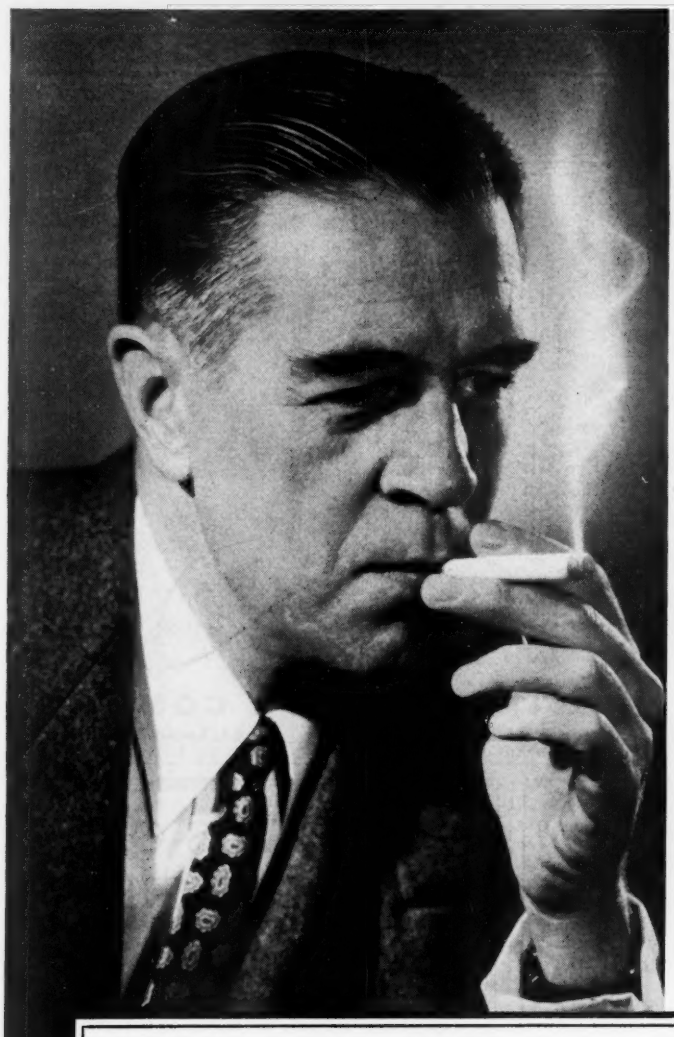

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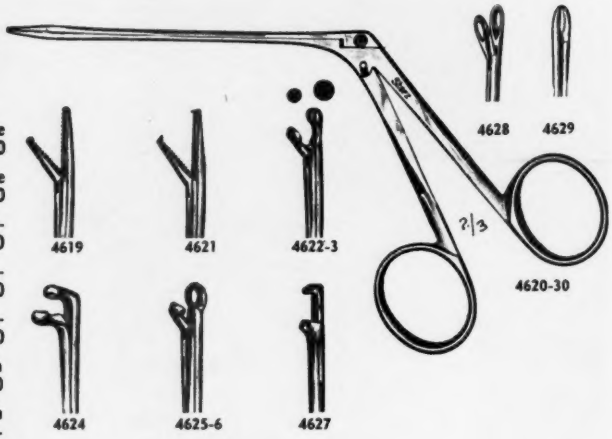

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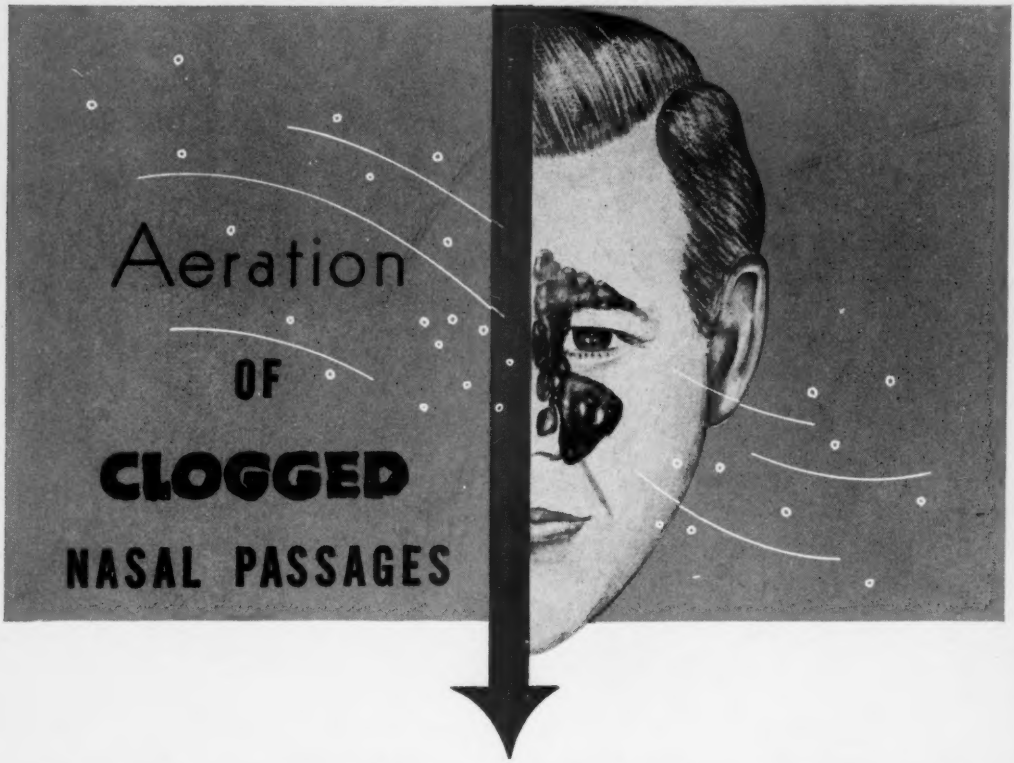

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1. Van Alyea, O. E., and Donnelly, Allen: Arch. Otolaryng., 49:234, Feb., 1949.

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